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PRACTICAL DIAGNOSIS:

THE USE OF

SYMPTOMS AND PHYSICAL SIGNS IN THE DIAGNOSIS OF DISEASE.

FIFTH EDITION, REVISED AND ENLARGED.

BY

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TICAL THERAPEUTICS.

ILLUSTRATED WITH 236 ENGRAVINGS AND 25 PLATES.

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1902

THIS BOOK

IS

DEDICATED TO MY FRIEND AND COLLEAGUE,

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32067

PREFACE TO THE FIFTH EDITION.

IN preparing the fifth edition of this book the author has rewritten a very large part of it, and has endeavored by the addition of much new material and by the careful revision of the earlier text to keep the book abreast of the advances in diagnostic technique, with the object of providing the practitioner with a manual which will be an aid to him in studying his cases. As pointed out in the preface to the first edition, the primary object of the work is to present the symptoms of a disease as they appear, and from this group of symptoms to arrive at a diagnosis, following the methods which are ordinarily employed at the bedside. Thus, if a patient has paraplegia, the reader will find in the chapter on the Feet and Legs a description of the symptoms of the various causes of this loss of power in the lower limbs, and so be led to a diagnosis of locomotor ataxia, myelitis, or neuritis, as the case may be, and in the chapter on Vomiting there will be found discussed the various causes which produce this annoying and dangerous symptom.

The present edition differs materially from its predecessors in the fact that its scope has been broadened to include not only the symptoms, discussed in the manner just described, but also the physical signs and clinical tests which experience has proved to be reliable. These have been considered much more fully than before, with the object of making the book as complete as possible. A large number of illustrations, most of them dealing with actual cases, have been introduced. The author wishes to express his appreciation of the cordial reception given to this work in its earlier editions, and to bespeak for it an equally favorable acceptance in its present amplified form, the more so as it appears simultaneously with the ninth edition of his Text-book of Practical Therapeutics, to which it has always been a companion volume.

H. A. H.

N. W. COR. SPRUCE AND EIGHTEENTH STS.,
August, 1902.

PREFACE TO THE FIRST EDITION.

THE object of this volume is to place before the physician and student the subject of medical diagnosis as it is met at the bedside. To accomplish this the symptoms used in diagnosis are discussed first, and their application to determine the character of the disease follows. Thus, instead of describing locomotor ataxia or myelitis, there will be found in the chapter on the Feet and Legs a discussion of the various forms of and causes of paraplegia, so that a physician who is consulted by a paraplegic patient can in a few moments find the various causes of this condition and the differential diagnosis between each. So, in the chapter on the Tongue, its appearance in disease, both local and remote, is discussed. In other words, this book is written upon a plan quite the reverse of that commonly followed, for in the ordinary treatises on diagnosis the physician is forced to make a supposititious diagnosis, and, having done this, turn to his reference book and read the article dealing with the disease supposed to be present, when if the description fails to coincide with the symptoms of his case he must make another guess and read another article. In this book, however, the discovery of any marked symptom will lead directly to the diagnosis. Thus, if the patient is vomiting, in the chapter on Vomiting will be found its various causes and its diagnostic significance, and the differentiation of each form of this affection from another.

The value of the book is increased by the preparation of two indexes: one of symptoms and the other of diseases.

Basing his efforts upon the experience which he has had in both didactic and clinical teaching of large classes of students during the last twelve years, the author hopes that the work may in some degree lighten the labors of the general practitioner and student, and relieve the all-important subject of diagnosis of some of the difficulties which surround it. He has also endeavored to make the text serve as an aid to the rational use of his *Text-book of Practical Therapeutics.*

PHILADELPHIA, 222 SOUTH FIFTEENTH STREET,
August, 1896.

CONTENTS.

INTRODUCTION.

GENERAL DIAGNOSTIC CONSIDERATIONS.

PART I.

THE MANIFESTATION OF DISEASE IN ORGANS.

CHAPTER I.

THE FACE AND HEAD.

PAGE

The expression and color of the face—Facial deformity—Facial paralysis, unilateral and bilateral—Ptosis—Facial spasm—The shape of the head—The movements and position of the head 27

CHAPTER II.

THE HANDS AND ARMS.

The general appearance of the hands and arms—The shape of the hands in disease—Spasms of the fingers—Tremors of the hands—Paralysis of the hands and arms 58

CHAPTER III.

THE FEET AND LEGS.

The general appearance of the feet and legs when clothed—The gait—Spastic paraplegia—Paraplegia without spastic contraction—Crural monoplegia—Deformities of the feet and legs—The joints—Alterations in the nutrition of the feet and legs aside from a change in the muscles 90

CHAPTER IV.

HEMIPLEGIA 136

CHAPTER V.

THE TONGUE, MOUTH, PHARYNX, AND ŒSOPHAGUS.

PAGE

The general appearance of the tongue—Its coating—Its appearance in poisoning—Fissures and ulcers of the tongue—Eruptions on the tongue—Atrophy and hypertrophy of the tongue—Paralysis—Tremor and spasm of the tongue—Tonsillitis—Diphtheria—Pharyngitis—Diseases of the œsophagus 150

CHAPTER VI.

THE SKIN.

The color of the skin—Eruptions on the skin—Gangrene, ulcers, and sloughs—Scars, sweating, dryness, œdema, hardness—Anæsthesia and hemianæsthesia—Paræsthesia, hyperæsthesia, itching . . 169

CHAPTER VII.

THE THORAX AND ITS VISCERA.

The inspection of the normal and abnormal chest—Their topography—Alterations in the shape of the thorax—The rhythm of the respirations—The results of using inspection, palpation, percussion, and auscultation in health and disease—The characteristic signs and symptoms of the various diseases of the thoracic organs . . 241

CHAPTER VIII.

THE ABDOMEN AND THE ABDOMINAL VISCERA.

The surface of the abdomen—Changes in the appearance and shape of the abdominal wall—The signs and symptoms of disease of the abdominal organs 320

CHAPTER IX.

THE BLOODVESSELS AND PULSE.

The condition of the bloodvessels on palpation—Feeling and counting the pulse—The quality, force, and volume of the pulse in health and disease 359

CHAPTER X.

THE BLOOD.

The various forms of red and white corpuscles—Their proportionate number in health and disease—Alterations in their form and character—The hæmoglobin of the blood in health and disease—The various forms of anæmia—Leukæmia and pseudo-leukæmia—Parasites of the blood 367

CHAPTER XI.

THE URINARY BLADDER AND THE URINE.

PAGE

Disorders and diseases of the urinary bladder—Retention of urine—Incontinence of urine—The characteristics of normal and abnormal urine—The normal and abnormal contents of the urine—Their significance—Tests for the contents of the urine . . . 402

CHAPTER XII.

THE BOWELS AND FECES.

Constipation and diarrhœa—The cause of these two symptoms and their diagnosis —The diseases in which these symptoms occur—Choleraic diarrhœa—Dysentery—The color of the feces—Intestinal parasites 449

CHAPTER XIII.

THE EYE.

The general diagnostic indications afforded by the eye—Diplopia and disorder of the external ocular muscles—Strabismus and squint—Disorder of the internal ocular muscles—The pupil—Hemianopsia —The visual fields—Color vision—The optic nerve and its lesions —Retinitis—Amblyopia and blindness 464

PART II.

THE MANIFESTATION OF DISEASE BY SYMPTOMS.

CHAPTER I.

CHILLS, FEVER, AND SUBNORMAL TEMPERATURES.

The methods of taking the temperature—The significance of fever—The febrile movements of various diseases 501

CHAPTER II.

HEADACHE AND VERTIGO.

The causes of headache—Digestive headache—Headaches due to the eyes—Headaches due to cerebral tumor and abscess—Headaches due to syphilis—Headaches complicating acute diseases . . . 531

CHAPTER III.

PAGE
COMA OR UNCONSCIOUSNESS 548

CHAPTER IV.

CONVULSIONS OR GENERAL SPASMS.

The convulsions of epilepsy in its various forms—Of infancy—Of hysteria—Tetanic convulsions—Spasms—Chorea 556

CHAPTER V.

HICCOUGH, VOMITING, REGURGITATION, AND THE CHARACTER OF THE VOMIT—DYSPHAGIA.

Due to uræmia—Cerebral lesions—Intestinal obstruction—Peritonitis—Cholera—Gastric disease – Hepatic disease—Poisons—The appearance of vomit 576

CHAPTER VI.

COUGH AND EXPECTORATION.

The varieties of and indications of cough—The causes of cough—The sputum—Its pathological significance 596

CHAPTER VII.

PAIN.

The kinds of pain—The significance of its locality—Colic . . . 609

CHAPTER VIII.

TENDON-REFLEXES AND MUSCLE-TONE.

The knee-jerk and ankle-clonus—The arm-jerk—The significance of decreased and increased reflexes 625

CHAPTER IX.

SPEECH.

The changes in the speech and voice—Their significance—Aphasia—Apraxia—Alexia—Paraphasia 631

PRACTICAL DIAGNOSIS.

INTRODUCTION.

GENERAL DIAGNOSTIC CONSIDERATIONS.

A CLEAR understanding by the physician of the value of the symptoms of disease which he sees and of those described by the patient is of vital importance for the purpose of diagnosis and treatment, and one of the advantages of older physicians over their younger brethren is the ability which they have gained through long training to grasp the essential details of a case almost at their first glance at the patient. Much of this ability is unconsciously possessed because it is gained by a gradual process, yet it is none the less valuable, and its possession often impresses the patient with the insight which his physician has into his case. At first it is impossible for the novice to cast aside the minor symptoms, which the patient emphasizes as his major ones, and to perceive clearly that one or two facts that have been belittled in the narration of the story of the illness are in reality the stalk about which everything else in the case must be made to cluster.

While the physician should train his eye to take note of all variations from the normal which are shown by the patient, he must be careful not to permit himself to depend for his diagnosis on these signs alone nor upon those described by the sick man. On the contrary, he should carefully train his senses of touch, hearing, sight, and discover the so-called physical signs which can be elicited only by auscultation, percussion, palpation, and mensuration. The objective symptoms seen by the physician, the subjective symptoms described by the patient, and the so-called physical signs elicited by the methods just named, are to be joined together in forming a diagnostic view of the case. Unless this combination is made, a faulty diagnosis will often be reached. In many instances chemical and microscopical examinations of the blood, the urine, the feces, and even of the tissues themselves, may be needful to determine in a given case what the ailment may be.

Again, it is never to be forgotten that negative signs are as important as positive signs in many cases. The absence of a heart murmur in a case of failing circulation is not a sign that the heart valves are healthy, but that the heart is too feeble to make a murmur, and the absence of râles or clearly heard breath-sounds in a lung may be due to engorgement without exudate instead of indicating that the lung is not diseased.

Let us suppose the patient before the physician is one who has been able to walk into the office or dispensary. The attentive physician can at once gather much information about the case from the clothing, the gait, the build, the voice, the expression, and the manner. The thin man, with a peaked face and provided with an unusually warm overcoat, and still further wrapped up with a muffler almost to his eyes, is in all probability a sufferer from some pulmonary or throat difficulty, while the heavily built, phlegmatic individual, with a large head and well-filled paunch, is much more apt to suffer from gastro-intestinal or biliary catarrhs. Such a person will probably be one who habitually wears his coat open on the coldest days. Again, chronic drunkards, or persons whose mental powers are failing, often are exceedingly careless about their clothing, buttoning the coat or the trowsers with the wrong buttons, and keeping the garments dirty and spotted. Some cases of diabetes have first been discovered by the white spots on the trowsers, as the result of having allowed a few drops of urine to fall on the cloth, where they have dried. Old men who have incontinence of urine often wear trowsers which are stained in front, and they often have an ammoniacal odor about them from this cause.

The various forms of gait which indicate actual disease will be found discussed in the chapter on the legs and feet, but it may be mentioned in passing that, in addition to these changes, which are dependent upon actual disease of the legs or the nervous system supplying them, the bearing and stride of a patient will often give us a clear idea of his general tone. The neurasthenic patient walks feebly or with a step that might be called ataxic, while the strong, hearty man of good physique strides along with a gait quite different from this, or from that of an individual who is delicate and feeble.

Similarly, the patient's build betokens disease or health. The thin, tall, and hollow-chested person is recognized as a fair mark for the tubercle-bacillus, and the heavy, closely knit, phlegmatic man as one who may suffer from hepatic disorders. Again, the bearing of a person possessing a highly organized nervous system shows itself in the constant activity of his mind and body. No part is quiet for more than a moment, and drugs are more apt to produce extraordinary symptoms as the result of idiosyncrasies in this type of patient than in any other.

The breath of the patient may call the physician's attention to some important facts in connection with the case. If it is sweet or vinous in odor, this may be due to diabetes mellitus, or if it be ammoniacal or urinous, uræmia may be present. The previous use of bismuth may give it a garlic-like smell, due to the contaminating tellurium, and various aromatic or volatile drugs, such as turpentine or copaiba, may be eliminated in the breath. In cases of advanced bronchiectasis the breath is often foul, and is insupportably so in gangrene of the lung or when an empyema has ruptured into a bronchus. It is similarly offensive in gangrenous stomatitis, and often very disagreeable in tonsillitis. In diphtheria it has a peculiarly sickening and sweetish odor. When the odor is not dependent on these causes it may be due to ozæna, or chronic atrophic nasal catarrh, to the presence of decaying food between the teeth or secretions in the crypts of the tonsils. Moderate fetor is usually due to disordered digestion and constipation.

When the physician has gathered as much information as possible as to the age and general condition of his patient, by a careful scrutiny of his face and extremities, of which scrutiny, however, the patient should be unconscious, he should ask him to tell what brings him for advice, and, as a rule, this will be the opportunity the sufferer seeks to pour out the story of his ailment as he sees or feels it. Often the story will seem wearisome, and, to the educated mind of the physician, wandering or unnecessary; but to the patient every word seems of the greatest importance, and to show any lack of interest may give the impression of carelessness, or it may interrupt the story just as a most important symptom is about to be described. Even if the patient is unable to convey in words a very clear idea of his condition, the manner in which his story is told, the character of his speech, and the expression of his face while speaking may give useful information as to his ailment or general state.

If, instead of the patient being an office or dispensary case, he is one who is being visited at home, the fact that the patient meets the physician in one of the living rooms rather than in a bedroom indicates either that there is little immediate danger in the case, or, at least, that the difficulty is not acute, but chronic in type, as some slowly progressing form of pulmonary, cardiac, or renal disease. Of course, there are exceptions to this rule, as in the case of a patient who, having caught a heavy cold, is remaining indoors, but not in bed, for prudence' sake. Or, again, if on seeing the patient we find him sitting in a chair only partly dressed and propped up with pillows, or instead leaning forward upon the back of a chair placed in front of him, we know that he is the subject most probably of an acute or chronic heart disease, most likely an acute exacerbation of the latter. A glance at the face of such a patient, revealing a trembling nostril, blue lips, or an anxious facies, will aid still further in directing attention to the heart or lungs, and the hands if examined will appear relaxed and livid or darkened in hue, indicating capillary stasis and deficient oxidation in the blood. In other cases, however, the patient found sitting propped up with pillows may be a convalescent from some long illness; but if so, the general atmosphere of the patient is better, and the surroundings are apt to be more tidy.

If we find the patient in bed, he may be lying abnormally quiet as the result of faintness or acute nausea, or, perhaps, from partial or complete coma due to cerebral or renal disease, or from the effects of some drug; or, again, he may be rolling about the bed from the pain of acute belly-ache, or be keeping his legs and body very still while his hands and head are ever on the move to prevent anything from suddenly approaching or touching his abdominal wall, as in peritonitis. The striking difference between the activity of the head and the fixation of the lower part of the body, in peritonitis, is notable. Sometimes, however, anxious restlessness indicates acute internal or external hemorrhage; but here the movements are minute though active, and the patient does not expend so much strength as he does when suffering from pain. In acute articular rheumatism the patient lies, if many of his joints are involved, in a peculiarly helpless position as if he were glued fast to the bed although the active movement of the eyeballs as they follow persons moving about the room gives quite a different aspect from that of a patient immovable from stupor. Usually a patient who is lying on his side turns on his back as the physician or nurse approaches, in order to face his visitor; but if he persistently remain on the side without moving, except partly to turn his head, we may suspect that in that posture he is most comfortable, and that the position is assumed for its comfort or to relieve pain or dyspnœa, which are suffered from when another attitude is taken. Thus, in acute pleurisy the patient lies with the affected side uppermost, because it is too sore to permit him to touch it to the bed; whereas if the stage of effusion has arrived, he lies on the affected side, in order to give the side which is healthy free play in compensatory respiratory movements, and to remove the pressure of the effusion from the healthy lung. If the patient lying in this posture is not suffering from pleurisy, his position may be due to an effort to relieve the discomfort caused by an enlarged liver. The fact that the patient lies constantly on the back is also a characteristic of grave and advanced disease in some instances. Very ill persons almost never lie on the side, and the fact that a desperately ill case of yesterday is found lying on the side to-day is encouraging. Persons with severe heart disease, emphysema, or chronic bronchitis, are rarely, if ever, able to lie prone in bed, and have to be more or less propped up with bed-rests and pillows. Large growths in the abdominal cavity producing pressure on the diaphragm also necessitate this semiprone posture, and double pleural effusions, or pulmonary consolidation or œdema, require the upright or half-reclining attitude in order that the upper parts of the lung may be used to advantage.

In asthma the patient frequently is found sitting up in bed with the arms reaching back of the hips as props to fix the chest and to hold the body erect. So too in advanced cardiac and renal disease the patient will often take a seat in a chair and rest the head and hands on the back of another chair placed in front of him, both eating and sleeping in this attitude.

Again, if the patient wakes when spoken to and then drops off to sleep at once, some form of poisoning may be present, as from opium, or the poisoning of advanced hepatic or renal cirrhosis may be present.

(For the significance of picking at the bedclothes, see chapter on the Hands and Arms.)

We can next pass to a consideration of the objects to be sought in questioning a patient as to the illness from which he is suffering. Often much information can be gained by a well-directed question, and a favorable impression can be made upon the patient by the manner in which it is put and the bearing which it has on his case. Thus, if a man is evidently much emaciated and his clothes fit him loosely, a question in regard to his loss of flesh is very appropriate; but if he is manifestly too stout for comfort such a question will be most unfortunate. Or, again, if a young married woman comes complaining of constant sickness of the stomach and a fanciful appetite, and the physician directs all his questions to the condition of the stomach without an eye to a slight increase in size about the waist or below it, his professional acumen is in grave danger of being libelled by that same woman, who knows, or soon finds out, that her discomfort is due to pregnancy.

If the woman is unmarried and there is no evidence of gastric disorder on her tongue, it is well to remember what Battey, of Georgia, said in regard to this condition: "Always believe a young unmarried woman with abdominal tumor, of high social position and unimpeachable virtue, if she has been watched over by a platonic and abstemious young cousin of the male persuasion while the mother went out, to be pregnant."

Again, if a married woman of some years tells her physician that she has no children, the physician naturally asks some questions which elicit the fact that she has had frequent miscarriages. He in this way finds out quite as much about probable syphilitic infection as if the question had been put: "Have you ever had a sore on your privates?" which would embarrass the patient, produce domestic troubles, and probably be lied about if she was forced to answer the question.

Again, when asking a woman about the health of the living parents, or the cause of death of the dead, care should be taken not to ask a direct question, as, for example, whether the mother has died of cancer, for the patient may be already greatly worried lest she has that disease. It is better to ask the cause of death, or of the illness she is suffering from. If the story is that the parents died of "bronchitis," in all human probability the real cause of death was tuberculosis of the lungs.

If the patient complains of pain, past or present, the best way in which to discover its true seat is to ask him to place his hand on the affected part, as in this way errors in his description of his anatomy will not be committed, and false impressions will not be conveyed to the physician's mind. Even this direct method of showing the area of pain is not to be absolutely relied upon, for often pains are referred to parts in which there is no disease. Thus, the pain of coxalgia is apt to be felt in the knee and ankle, and in children the pain of acute pulmonary disease is often described by the patient as felt in the abdomen. If the pain has been really abdominal, there will, in many cases, have been diarrhœa or free passage of flatus. It is not to be forgotten, on the other hand, that a question which discovers the fact of several movements of the bowels does not prove the presence of true diarrhœa, because a purgative may have been taken by the patient.

In asking questions as to constipation the physician must not forget that the opinion of the patient as to what constitutes regularity of bowel-movement is of very little value in many instances. A daily movement is not known to many patients, and a movement every few days may be quite sufficient to justify the statement, in their opinion, that no constipation is present.

The young physician, in particular, in asking questions of women patients of the better class, should not hesitate to ask direct questions as to the state of the bowels or of the menstrual function. To hesitate or ask indirect questions about such matters simply produces embarrassment which otherwise would not exist, and intimates that the question is one of doubtful propriety, when in reality it is most important and proper.

If the patient to be examined is a child, it is well for the physician to remember that his mere presence as a stranger may be a source of alarm, and that the association in the child's mind of sickness and the doctor, and badly tasting medicines, is sufficient to render him a much-to-be-dreaded individual. Generally it is best, on entering the room where the child is, to pretend to pay no attention to it whatever, but to engage in conversation with the mother or other person, speaking of the case in a way which the child will not understand. Very often this very lack of attention will result in the child forcing the recognition of his presence upon the physician by making the first advances toward friendship, and this is particularly apt to be the case if the child is already spoiled by over-attention by the family and friends. Time should always be given the child to grow accustomed to the peculiarities of the visitor, and, if any instrument for diagnosis is to be employed, it is best to hold it in the hand as if it were a plaything before attempting to put it into actual use. The tact which the physician must exercise in diverting a sick child is an essential to the successful treatment of children. Some physicians are welcomed to a house by the sick and well as a Santa Claus would be, and others, devoid of the trait of amusing children, are fled from as if they were dragons.

During the time that the physician is allowing the child to get accustomed to his presence he should be gaining much useful information about the case by observing the movements and expressions of the child; its color, size, nutrition, breathing; the shape and size of its head; the condition of its lips, whether moist or dry, red, livid, or pale; and, if the child is speaking, the tone of its voice, or, if crying, the character of its cry. It is needless to state that a child may cry from fright, from pain, anger, or hunger. Constant screaming crying is, however, nearly always due to the pain of earache or hunger, for abdominal colic is usually intermittent. If there be pain in the ear, the hand will often be rubbed over the affected side of the head, and the child will not be pacified by the offer of the breast. If the child coughs, and then begins to cry, pneumonia or pleurisy may be present; or in other cases the pain is so great that the child is cryless. A sharp, piercing shriek of crying indicates the pain of earache or of meningitis in many cases.

If the crying child be placed at the breast, which it takes with avidity only to drop the nipple in a moment with a cry of pain or anger, one of several conditions is present: either the child has stomatitis or the breast is empty; or, again, if it seizes the breast and then lets go with a gasp, it probably has coryza or syphilitic snuffles, which prevents it from breathing through the nose while sucking. Similar signs may be present in any other condition producing shortness of breath.

If a child over four months of age cries and sheds no tears in the course of an illness, this is an unfavorable sign.

It is important to notice whether there is languor or a tendency to play. A healthy infant, when awake and well fed, is always kicking and cooing and moving its arms about, and has a happy expression on its face; whereas if any cerebral trouble is present, it often has an anxious frown, or its hands are placed on the side of its head or rubbed over the vertex.

In a perfectly healthy child which is sleeping the respiration should be practically inaudible, and it is a good practice to note the regularity of the breathing in all patients while they are asleep, as it is then unaffected by voluntary effort. In children a sighing breathing, or one disturbed in rhythm, often indicates a disturbed digestion or fever.

The physician should always, by careful questioning of the nurse or mother, find out how long the illness has lasted, the manner in which it began, the fact as to whether a similar attack has occurred before in this or other children of the family, and the state of the temper, appetite, bowels, and urine of the patient, for an irritable temper in a child means ill health, as does also a poor appetite, constipation, diarrhœa, or abnormal urine.

The expression of the face, shape of the head, and similar noteworthy points in the diagnosis of the case will be more thoroughly discussed in the chapter devoted to these parts.

When it comes to a close examination of the child, great care must be exercised. The character and rapidity of the respirations are best studied at a distance before excitement has disturbed them, and the best time for listening to a young child's chest is when it is held over the shoulder of the mother as if she were carrying it for a walk, or, if the child can be taken in the physician's arms, its buttocks should rest on one hand, while its chest leans against the other. In this way the physician can listen to the back of the chest without difficulty, keeping the child amused by walking up and down the room while it is in his hands.

If it is not possible by any bribe to cause the child to protrude the tongue for examination, the physician will often be able to see this organ when the mouth is widely opened in crying.

In taking a child's pulse it is best to take it while it is asleep, if possible, as the excitement of the physician's visit or the crying on awakening will greatly increase the pulse-rate.

PART I.

THE MANIFESTATION OF DISEASE IN ORGANS.

CHAPTER I.

THE FACE AND HEAD.

The expression and color of the face—Facial deformity—Facial paralysis, unilateral and bilateral—Ptosis—Facial spasm—The shape of the head—The movements and position of the head and neck.

So much can be learned by the physician from the expression and general appearance of a patient's face and the carriage and shape of his head, that a careful inspection of these parts should always be made. For this reason, in the consulting-room and at the bedside, the physician should always arrange his chair in such a way that the light falls upon the face of his patient, while his own is in the shadow, and this is of importance not only because the facial expression of the patient can thus be well seen, but also because it prevents the patient from making a too close scrutiny of the physician's face with the object of detecting encouragement, lack of sympathy, or alarm.

The Face.

The Expression is produced by the formation of creases, or alterations in the contour of the skin and subcutaneous tissues by trophic and muscular action, and these changes are in time brought about by the mental tendencies and habits of the patient, his temperament, his intellectual development, his exposure to outdoor or indoor influences, and finally, and these are very important, by pathological processes which may be going on somewhere in his body. The temper of the man also affects his expression, particularly as he approaches middle life, and he looks amiable, capable of sudden anger, or sullen, as the case may be.

The intellectual face is easily recognized. Sometimes it is deeply thoughtful and placid, at others eager or keenly alive to the surroundings or the conversation, and it separates the man descended from several generations of men who have lived as thinkers from him whose ancestors have been but recently wage-earners by physical labor, involving only ordinary human intelligence.

Fulness of the lips, particularly of the lower lip, is supposed to be present in persons of strong sexual appetite, and often indicates a phlegmatic temperament, whereas the thin, mobile lip is typical of the high-strung, nervous individual.

The expression of the lips as a whole is also to be regarded in connection with the expression of smiling. The risus sardonicus of strychnine-poisoning or tetanus is quite characteristic, and the simple smile of hysteria is equally notorious.

The skin of the face and the expression about the eyes of one who has been exposed for years to the weather are so characteristic as to need no description, while the face of the clerk, whose life is almost entirely spent indoors, is pale and wan.

Similarly, the face of a person who uses alcohol to excess is generally flushed, heavy, and more or less expressionless. The eyelids are reddened more than normal, and the skin is apt to be puffy and unhealthy-looking. Women at the menstrual period, or when suffering from menstrual disorders, often have dark areas under the eyes, and pigmentation of the eyelids is often seen very early in pregnancy. In women, and sometimes in men, excessive fatigue and loss of sleep cause marked infra-orbital discolorations. A puffiness under the eyes, most noticeable in the morning, may indicate renal lesions or the excessive use of arsenic, or if it is unilateral it probably depends upon some local inflammation of the eye or rarely upon disease of one of the cerebral sinuses. So, too, an ecchymotic spot under the eye may be due to a bruise, to some one of the forms of purpura, or to scurvy.

The color of the face is discussed in the chapter on the Skin, but it is not out of place to note at this point the pallor of the face in fright, faintness from hemorrhage, acute or chronic, that due to lack of proper food, and the peculiar pallor of chlorosis. In the latter disease the faint yellowish-green tinge of the skin in some parts of the face, which still retains its plumpness, is quite typical. A parchment-like skin stretched over the face so that it appears as if dried over the under-structures is seen in some young persons suffering from syphilis, and in some cases of alcoholic hepatic cirrhosis.

The color of the face may be rendered gray or bluish by the ingestion of overdoses of the coal-tar products, such as acetanilid, antipyrin, and phenacetin, and it is curious that this effect is best seen when the patient is viewed at a little distance.

(For the indications of facial cyanosis, see chapter on the Skin.)

In view of the extraordinary variations seen in the expression of the face in the healthy it is not surprising that this part of the body should give the physician, when studying disease, so much useful information. It is an interesting fact, too, and one not unworthy of note, that the true facial expression of a disease is rarely aped by a malingerer, and in all diseases is unrecognized by the patient even though he sees himself several times daily in a looking-glass. Thus it is by no means uncommon to see a person who is suffering from the onset of some sudden and grave disease bearing upon his face what we call "an expression of anxiety," when he himself as yet has no conception of the gravity of his illness. This expression is very characteristic of serious illness, and, though difficult to describe, when recognized becomes quite valuable as a diagnostic factor, particularly as it rarely, if ever, is exaggerated by the patient who bears it. It is seen most markedly in cases of severe acute croupous pneumonia or peritonitis, or after severe injuries.

When persons have had continuous pain for a long time, as in patients who have growths of a malignant character or other organic disease, the expression of the face, naturally gentle, often becomes hard and stony, or, if the pain be in the head, the expression is not only that of pain, but of profound mental depression. In cases of carcinoma the face becomes thin, its skin yellow and straw-colored, and oftentimes greasy and thick, and there is often a marked expression of anxiety. On the other hand, the patient sometimes has a dogged expression on his face as if he had been told of the true cause of his illness, and was rebelling against the inevitable progress of the disease.

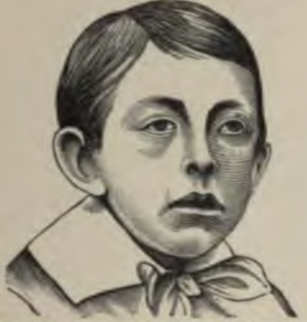
In the case of children, much information can be gained as to the state of the system by the facial expression, particularly while the child sleeps. If it is asleep and healthy and well, the eyelids are closed, the lips are never so slightly parted, the nostrils are practically immobile, and the general expression is very peaceful. If, on the other hand, the eyelids of a sleeping child are slightly parted so as to show the whites of the eyes, there is probably present some digestive or nervous disturbance, perhaps accompanied by moderate pain. If in the course of an illness the eyelids remain far enough apart to result in glazing of the conjunctiva from dryness, this is a sign of grave import. Again, twitching of the eyelids often indicates nervous irritation or the early stages of the convulsive state, and it is not uncommon for an expression to pass over the face of a child who, while sleeping, is suffering from pain, which begins as a smile and ends with a drawing-in of the corners of the mouth, an expression somewhat like that seen on the face of a waking child when it seems to be in doubt as to whether to laugh or to cry. Whether asleep or awake a child in pain, if not crying, has a pinched look about its nose and mouth, and sometimes some idea of the seat of the pain may be gained by the part of the face which is drawn. When pain is in the head, the forehead is apt to be wrinkled into a frown; if the nose is pinched and drawn, it is said to show that the pain is in the chest; and if the upper lip is raised, pain is probably felt in the belly.

Aside from these symptomatic manifestations, however, we find in the face of a child several evidences of important diathetic tendencies, or even hereditary diseases. Thus we see the light flaxen-haired, slimly built child with a refined, *spirituelle* face and transparent skin, whose temporal veins can be easily traced and whose expression is often thoughtful and deep. Such a child often comes of tubercular parents, and is frequently a victim of tuberculosis, in one of its rapid forms, as it approaches puberty. Or, again, the child is "stocky" and cheesy-looking, apparently solid and sturdy, but its features are heavy or perhaps even coarse, while its neck is thick and short. Such a child is often a victim of tubercular bone disease. In other instances, a square projecting forehead with faulty bone-development elsewhere indicates rickets, or an immense, bulging forehead with a wizened, puny face beneath shows hydrocephalic tendencies. Sometimes a broadness of the bridge of the nose or marked flatness of it indicates congenital syphilis. Such a child is often much wasted, its features pinched, and its lips thin, while the flattened nasal bridge is bluish, and its face is often that of a little old man, shrivelled and wrinkled. Mucous patches at the corners of the mouth or around the anus are often found in such cases, and, if found, confirm the diagnosis of infantile syphilis. Finally, in respect to facial expression in childhood, attention must be called to the "fish mouth," vacuous, and "nose-pinched" expression of those children who are "mouth-breathers" from nasal obstruction. (Fig. 1.) Great immobility of the lips and cheeks may be due to mucous patches or other ulcerations of the buccal mucous membrane; and if high fever is present, the presence of herpetic blisters about the lips points to the possible presence of croupous pneumonia in the child or adult.

The face of a patient with acute fever is apt to be red and flushed, and the eyes bright; and if the disease be distinctly infectious, as in some cases of pneumonia, tuberculosis, and acute articular rheumatism, it may be covered with sweat.

In adults the facial expression of many diseases is even more characteristic than it is in children. Thus, we see in acute pulmonary phthisis the widely opened eye, the hunted expression, the quivering nostrils, the red flush over the malar bones, the wasting and dryness of the hair and skin, and the eager or apathetic glance of the eye.

FIG. 1.



Boy, aged seven. Mouth-breather, from obstruction of the pharynx; open mouth; vacant expression; pinched nostrils; dull eyes; drooping eyelids; round shoulders. (HOOPER.)

In severe croupous pneumonia the flushed face, with a deeper red on one cheek than the other, the anxious expression, and the dilated nostril are noteworthy; and in the dyspnœa of heart disease the dilated nostril and constant opening of the mouth, as if seeking for air, with the facial pallor or cyanosis, are characteristic. Often, too, in chronic cardiac or pulmonary disease producing slight difficulty in respiration, the patient's lips are seen to be slightly parted and dry, and may appear somewhat cyanotic. In children suffering from lesions of the mitral valve of the heart it is very common for some blurring or indistinctness of the features to be present.

One of the most characteristic facial expressions that we meet with is that of typhoid fever or fevers of a typhoid type. The face is dull and expressionless; the teeth are covered with sordes, which become brown and blackish by exposure or by discoloration from medicines and foods; the lips are often moved in a low muttering delirium; and the whole appearance is that of apathy. Even when spoken to, the face of a patient suffering from enteric fever rarely lights up in response to the greeting.

Equally, if not more, characteristic is the facial expression of acute peritonitis. The upper lip is drawn up in such a way as to show the teeth, and the expression of anxiety and nervous unrest is well developed. Similarly in abdominal pain due to other causes than peritonitis there is often a twitching of the muscles of the lip and about the eye which is quite typical. This twitch is said by Fothergill to be peculiar to pain below the diaphragm, and he is also responsible for the statement that it is best seen in the face of the parturient woman in the second stage of labor.

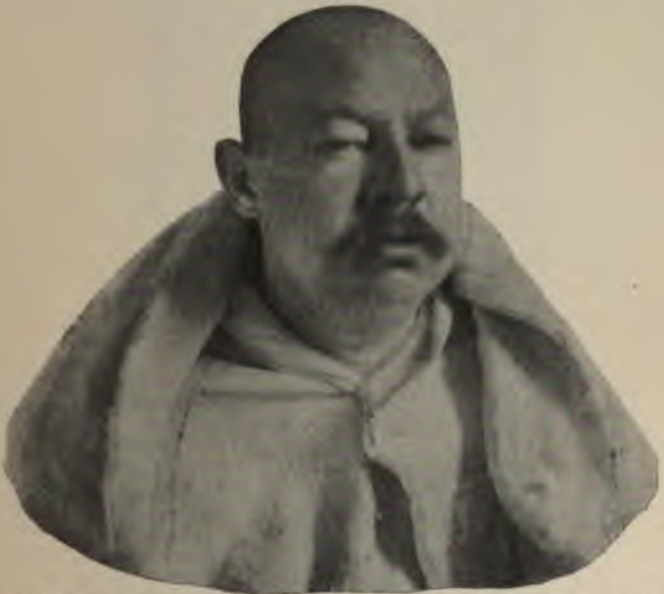
The facial expression of hysteria may be apathetic, or it is that of devotion, rage, or grief, and these expressions are fixed if the patient be cataleptic. If she is not cataleptic, not infrequently one expression may succeed the other, or in their place there comes that curious smile or vacuous expression of the face which is so characteristic. It should be remembered, however, that this vacant fatuous look may occur in women suffering from the early stages of disseminated sclerosis and in children with chorea. Then we have the elated facial expression of general paralysis of the insane, the excited look of acute mania, the beaten, weary, careworn look or apathetic glance of nervous exhaustion, and the hopeless expression of melancholia.

The face of paralysis agitans, sometimes called the "Parkinsonian visage," is distressed and pathetic, and yet somewhat intense. (See chapter on the Hands and Arms, and part of that on Tremor.)

A pale, puffy face, generally looking worn and weary, may be seen in cases of chronic or subacute renal disease. In children there is often in this condition a peculiar transparent or pearly look in the lower eyelid, so that it seems somewhat pellucid. Great swelling or œdema of the face is seen in erysipelas, dropsy (Fig. 2), and simple inflammatory swelling (see chapter on the Skin). In trichiniasis the eyelids are often swollen early in the disease, and then recover their normal appearance only to become swollen again later in the malady.

When the face bears a sleepy, listless expression, the forehead being devoid of wrinkles, and there are present faulty movements of the lips, which cannot be approximated, as in whistling, and at the same time the patient is unable to close the eyes entirely, although the lids droop, the physician should think of the possibility of these being the early symptoms of what has been called the "facio-humero-scapular" type of muscular atrophy (Landouzy and Déjèrine). The disease, as its name implies, speedily involves the scapulæ and arms after affecting the face, and exophthalmos is often present. This form of muscular atrophy lacks the fibrillary twitchings seen in spinal progressive muscular atrophy ; there are no changes in electrical excitability, except that owing to the loss of muscle-fibre the reaction is feeble. The facts that more than one member of the family is affected and that the disease is of long duration, added to these signs, render the diagnosis easy. It is a rare disease.

FIG. 2.



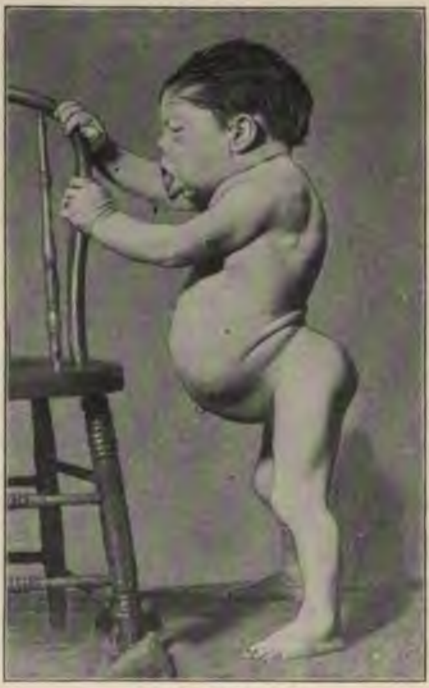
Face of a patient with general anasarca due to chronic parenchymatous nephritis. (From a patient in the author's wards—Jefferson Medical College Hospital.)

An appearance of the face almost identical with that just described is seen in Friedreich's ataxia, and is often one of the earlier manifestations of the disease; but the presence in Friedreich's ataxia of the ataxic gait, the jerky articulation, nystagmus, loss of knee-jerks, and absence of muscular atrophy separate it from the Landouzy-

Déjèrine type of muscular atrophy just described as facio-humero-scapular atrophy. (See Ataxia in chapter on Feet and Legs.)

The facial expression of cretinism is exceedingly characteristic. The nose is broad and flat, the eyelids are swollen, the lips greatly thickened, and the enlarged tongue lolls out of the mouth, from which saliva constantly dribbles, while the waxy skin and subnormal temperature of the body, with a poor circulation, slow respiration, and mental hebetude, complete the symptom-group. There is nearly always in well-developed cases marked lumbar lordosis. (Fig. 3.) The facial expression of myxœdema is heavy and listless, as a rule. (See page 37 and chapter on Skin.)

FIG. 3.



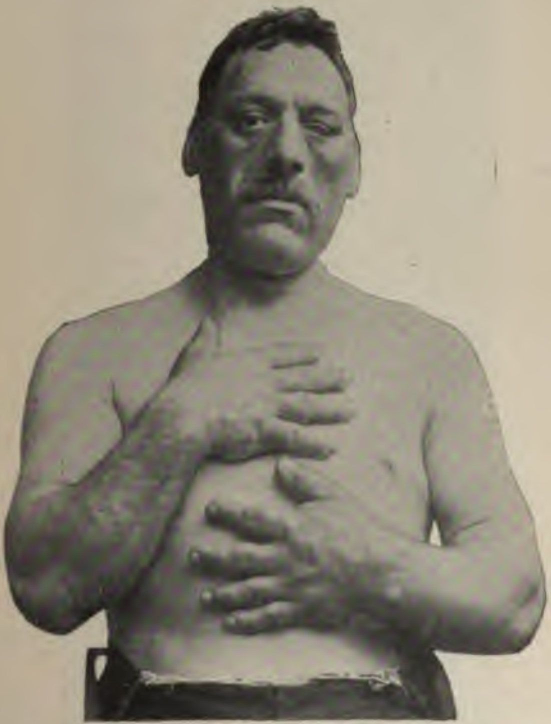
A cretin. (DERCUM.)

In certain forms of leprosy the face often becomes leonine, or lion-like in appearance.

The facies of exhausting disease about to produce death is very characteristic, and is seen frequently in cholera and in tuberculosis of the lungs or any state associated with profound collapse, such as internal hemorrhage. It is accompanied by pallor, cold extremities, and difficult breathing. This is called the "Hippocratic face," and is peculiar in the sinking-in of the temples where the jaw-muscles are inserted; the eyes are sunken, and around them are great hollows, so that the infra- and supra-orbital ridges become greatly accentuated. The eyelids are slightly parted, the cornea somewhat glazed; the nose pinched, its skin drawn; and the lower jaw somewhat dropped. Such a facial expression, if typical, is a sure forerunner of dissolution.

Facial Deformity. Facial asymmetry is sometimes seen as a congenital defect, and curiously enough is often developed in children who suffer from congenital wry-neck. This is not to be confused with that extraordinary affection called facial hemiatrophy, which usually begins in childhood in one spot, and slowly proceeds until one side of the face, sharply outlined from the other, becomes

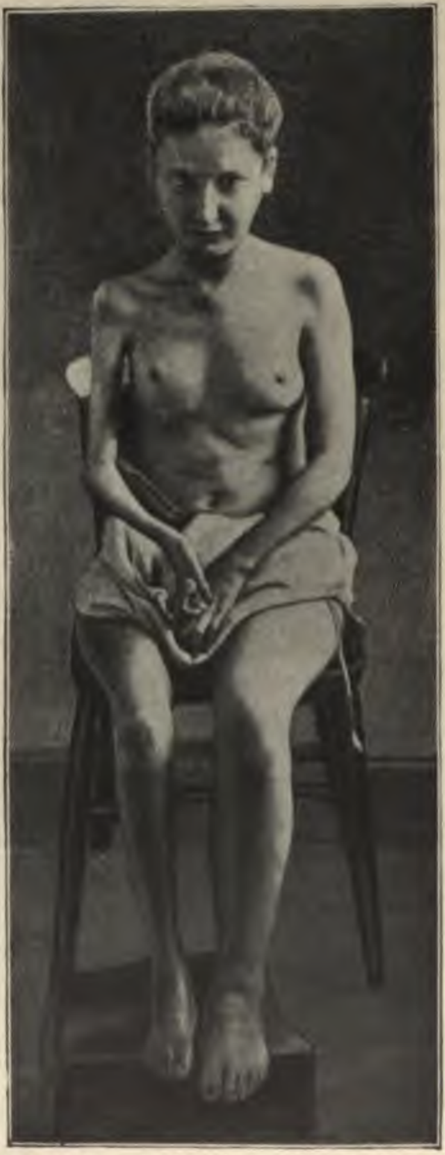
FIG. 4.



Acromegaly, showing the large face and hands. (DERCUM.)

wasted in its skin, muscles, bones, color, and hair. Even the eye may be sunken and shrunken. Rarely, this wasting extends over

FIG. 5.

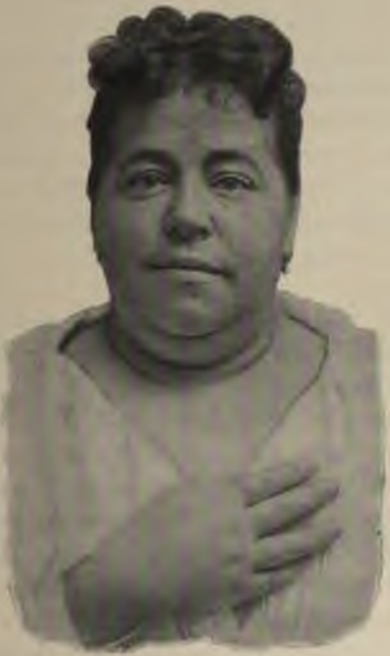


Martha F., showing atrophied condition of the right arm and leg as well as of the right side of the face and body. (STEVENS' case.)

the whole of one side of the body (see Fig. 5), and still more rarely is bilateral.

Sometimes in facial hemiatrophy the wasting is accompanied by painful twitchings, which increase with mental excitement. More rarely there is decrease in the acuity of taste and hearing on the affected side, while myosis, sweating, or excessive dryness of the skin may be also found on this side. Such symptoms as the last show involvement of the sympathetic nerve-fibres. The changes are probably due to disease of the fifth (trifacial) nerve.

FIG. 6.



Myxœdema. (MELTZER.)
The expression is usually more stupid and apathetic.

As to whether circumscribed scleroderma (morphœa) and facial hemiatrophy are identical—that is, whether the first is a well-developed form of the latter—is not decided. Hyde apparently regards them as identical. (See chapter on Skin, Scleroderma.)

Even more rare than facial hemiatrophy is facial hemihypertrophy, one side remaining normal in size and the other becoming gigantic.

The massive face of a person suffering from acromegaly is very characteristic. (Fig. 4.) The face has a full-moon broadness in myxœdema. (Fig. 6.) The enlargement of the bony parts of the skeleton, the kyphosis, and the comparative muscular feebleness of acromegaly aid in the diagnosis of that disease, for in myxœdema there is no true bony enlargement. The face in osteitis deformans is shaped like a triangle with the base upward. In osteitis deformans the shafts of the long bones become weakened, and their surfaces roughened from periosteal deposits. (See chapter on Hands and Arms.)

Unilateral Facial Paralysis. Very notable changes in the face are produced by paralysis, the palsy being, as a rule, unilateral and depending upon central or peripheral nerve lesions for its cause. Smiling, when unilateral paralysis is present, results in the drawing back of only one corner of the mouth (on the well side), and whistling or the pronunciation of labial sounds is difficult or impossible. The cheek of the paralyzed side is often puffed out with each expiration, but the wrinkling of the skin is on the side of the face which is not paralyzed, owing to contraction of the muscles which are unopposed.

(For a description of the general anatomy and physiology of the nervous tracts involved in paralysis of the face and elsewhere, see chapter on Hemiplegia.)

Unilateral paralysis is, as already stated, the form of facial paralysis most commonly seen, and is generally due to injury of the facial nerve-trunk. The lesion producing the paralysis may, however, be peripheral—that is, in the nerve itself—or central, that is, in the pons or the cerebral cortex. The former variety is the most common, provided the paralysis is purely facial, and it is usually due to inflammation of the nerve-sheath as it passes through the stylo-mastoid foramen, the loss of function being due to pressure on the axis-cylinders owing to the presence of swelling in so limited a canal. Such an attack will generally be found associated with a history of exposure to cold or injury by a blow, or with that of middle-ear disease with caries of the petrous portion of the temporal bone following otitis, which inflammatory process causes pressure on the nerve. It is not necessary for the otitis to be suppurative or for caries to exist in all cases, for it seems probable that by the extension of inflammation along the chorda tympani such a paralysis may result. If the disease be in the petrous portion of the temporal bone, in addition to paralysis of the muscles of expression there will also be loss of taste in the anterior part of the tongue due to involvement of the chorda tympani. The mouth is dry, owing to a lack of saliva, the salivary gland being paralyzed, and there may be deafness from paralysis of the stapedius muscle. Still more rarely facial paralysis results from swelling of the parotid gland or from a tumor in its neighborhood, and it may occur as the result of pressure by growths at the base of the brain, syphilitic or otherwise, from fracture of the base of the skull involving the petrous portion of the temporal bone, and very rarely, when the disease occurs in the newborn, from hemorrhage from the cerebellum during birth, or from pressure of forceps. (See below.) Finally, paralysis due to a peripheral lesion of the nerve may result from neuritis, and from primary hemorrhage into the nerve-sheath or into the stylo-mastoid canal. Facial paralysis may also arise from locomotor ataxia, the lesion being in the pons, and from hysteria. All these forms are very rare, comparatively speaking. The cerebral or medullary lesions which produce unilateral facial paralysis usually consist in the results of hemorrhage and tumor.

The determination that facial paralysis is due to a peripheral neuritis or pressure may be impossible at the first visit of the patient, if this visit is made, as it usually is, within a few hours of the onset of the malady; but the peripheral form separates itself from facial paralysis of cerebral origin in the course of ten days or two weeks, for, if the nerve is inflamed or pressed upon in the foramen, the muscles of the face speedily undergo degeneration, because they are cut off from their trophic centres. In the cerebral form, on the other hand, the trophic changes do not occur, and the reactions of degeneration fail to appear, because trophic impulses can still reach the facial nerve-trunk and the muscles which it supplies. In other words, electrical response in the paralyzed side remains normal in centric lesions and is lost in peripheral lesions. The only other conditions in which there can be developed the reaction of degeneration and the lesion not be in the nerve-trunk or foramen is when there is a tumor at the base of the brain involving the facial fibres below the facial nucleus or destroying the nucleus itself.

Very rarely in cerebral facial paralysis is the loss of power as complete as it is in the peripheral form. Again, in cerebral facial paralysis the eye on the paralyzed side can usually be closed and the forehead wrinkled, whereas in the peripheral form it cannot.

Why this should be so is not clear, unless it is that in the muscles used commonly in pairs, as in those of the forehead, there is an adequate nerve-supply through direct non-decussating tracts which innervate the muscles. When facial paralysis has associated with it none of the signs of peripheral wasting, and none of the remote causes of hemorrhage, embolism or thrombosis, such as result from impaired bloodvessels or a diseased heart, and when the paralysis comes on gradually (though it may be sudden from surrounding inflammation), the condition is probably due to cerebral tumor. This diagnosis is confirmed by the gradual spread of the paralysis to other parts, as the arm and then the leg on the same side of the body, and by the development, often before each additional spread of the paralysis, of a convulsion. The facial paralysis resulting from tumor at the base of the brain differs from that due to cerebral tumor or hemorrhage by the fact, already stated, that the reaction of degeneration quickly develops in the paralyzed part; that the parts supplied by the upper branch of the facial are often quite as much paralyzed as are those supplied by the lower branch, which is rare in the cerebral lesion; and there will commonly be found other evidences of a growth which, in a region so densely filled with important centres, speedily affects other functions. Thus, in association with this form of facial paralysis there will nearly always be found paralysis of the oculo-motor nerve, causing ptosis, a moderately dilated pupil, and external squint, and there may also be paralysis of the abducens or sixth nerve, which causes internal squint by paralysis of the external rectus muscle. (See Ptosis.) The optic nerve may show choked disk, and there may be disturbance of vision. (See chapter on Eye.) If the tumor is large, or is so placed as to involve the facial fibres for both sides as well as those of the oculo-motor, abducens, and optic nerves on both sides, all these symptoms become, of course, bilateral.

Facial palsy associated with deafness may indicate cerebellar tumor, the diagnosis of this cause being decided by the other cerebellar symptoms, such as the peculiar gait. (See chapter on Feet and Legs.) Such growths are not uncommon in children.

Sometimes very shortly after birth the child is seen to have a facial paralysis resulting from pressure by the forceps, which have slipped and injured the facial nerve, or have caused an extravasation of blood into the neighborhood of the parotid gland, thereby causing pressure on the nerve. The prognosis is usually favorable if due to such causes; but if the forceps have caused facial palsy by producing a cerebral hemorrhage, the outlook is bad.

The possibility of facial paralysis being due to hysteria should not be forgotten. The loss of power under these conditions may be unilateral or bilateral, generally the former. Its association with the symptoms of hysteria described in the chapter on the skin, and elsewhere in this book, will aid in making the diagnosis.

FIG. 7.

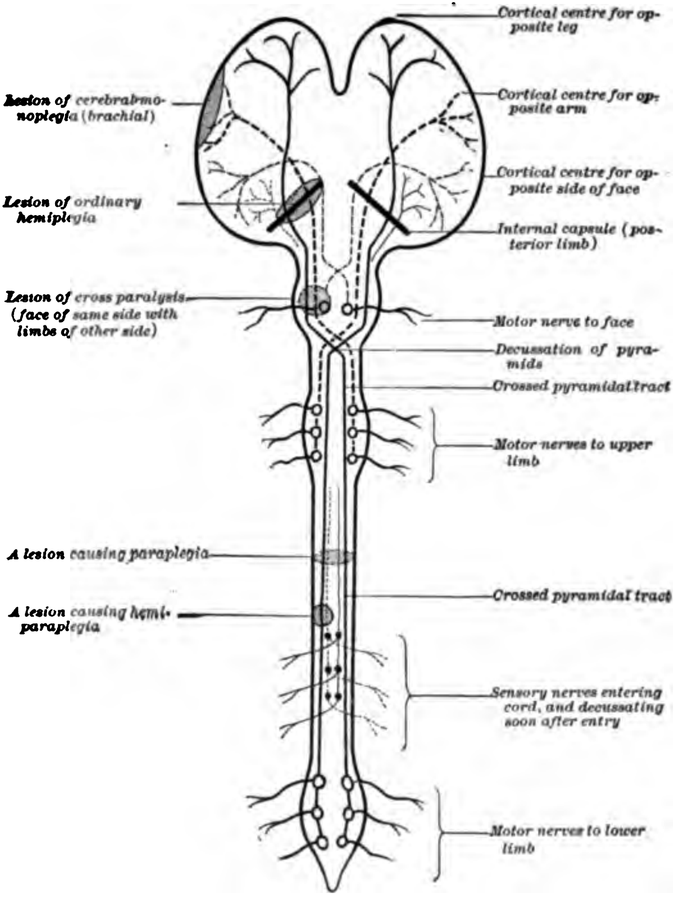

Cortical centre for opposite leg
Lesion of cerebral monoplegia (brachial)
Cortical centre for opposite arm
Cortical centre for opposite side of face
Lesion of ordinary hemiplegia
Internal capsule (posterior limb)
Lesion of cross paralysis (face of same side with limbs of other side)
Motor nerve to face
Decussation of pyramids
Crossed pyramidal tract
Motor nerves to upper limb
A lesion causing paraplegia
Crossed pyramidal tract
A lesion causing hemiparaplegia
Sensory nerves entering cord, and decussating soon after entry
Motor nerves to lower limb

Diagram to show the general arrangement of the motor tract and the effect of lesions at various points. (ORMEROD.)

There yet remain to be considered several forms of facial paralysis unilateral in character yet associated with paralysis elsewhere. These are as follows:

Unilateral facial paralysis very rarely occurs in association with monoplegia in acute anterior poliomyelitis. So seldom does it occur in this connection that it has been denied an existence. Often it is but temporary, while the monoplegia of the arm is permanent. It occurs more commonly with the disease in adults than in children.

Facial paralysis with arm paralysis of the same side, followed in a short time by paralysis of the leg of the opposite side, is quite a characteristic symptom of syphilitic arteritis at the base of the brain.

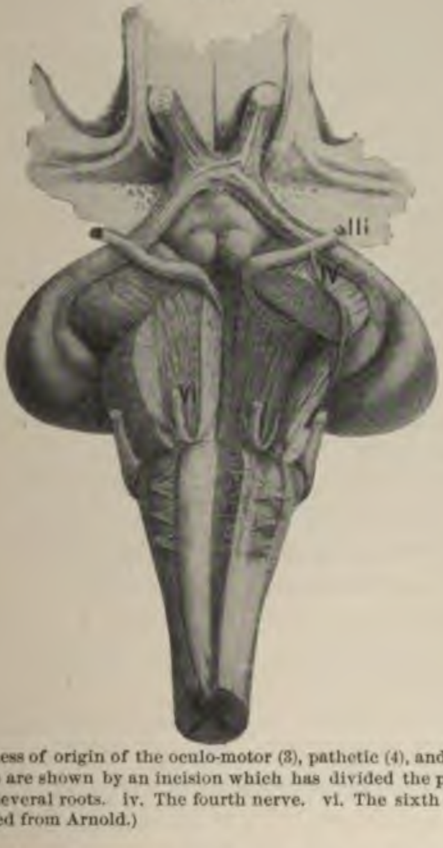
Crossed paralysis—that is, paralysis of the face on one side, and of the arm and leg on the other—is due to a lesion in the pons above the decussation of the pyramids and below that of the facial fibres. (Fig. 7.) Thus it is seen in this figure, on the left side, third inscription, that the lesion in the pons cuts off the motor fibres in the place indicated, thereby causing the distribution of the paralysis just named. (See also chapters on Hemiplegia and on Arms and Hands.)

Sometimes the muscles supplied by the facial nerve escape paralysis, but those of the jaw—namely, the masseters and temporals—become paralyzed either bilaterally or more commonly unilaterally. This is a rare affection, and depends upon paralysis of the inferior maxillary branch of the trifacial nerve. This may be due to pressure produced by growths or inflammatory processes at the base of the skull. It may also occur as the result of hemorrhage into the medulla, or from progressive bulbar paralysis.

Ptosis. In connection with the subject of facial paralysis that of ptosis, or drooping of the upper eyelid, must be considered. (Fig. 9.) It depends upon loss of function of the oculo-motor nerve or its nucleus. Ptosis is a symptom of the greatest importance, first, because it is so readily recognized; second, because it is a source of great annoyance and alarm to the patient; and third, and more important, it often aids us greatly in localizing lesions.

The presence of this symptom should call to the physician's mind the various causes which produce it. In the first place, it sometimes occurs as a congenital defect, and in such a case the history of the patient renders the diagnosis easy. If a congenital defect is not responsible for the ptosis, it must depend upon disease affecting the oculo-motor nerve itself or its nucleus. If the disease be sufficient to cause entire loss of function, we find, in addition to ptosis, that there is paralysis of all the external muscles of the eye except the superior oblique and external rectus, and in addition there will be a moderately dilated pupil, which will not contract, and paralysis of the ciliary muscle—that is, loss of accommodation. The eye can be moved outward by the action of the external rectus, and a little downward and inward by the superior oblique. Diplopia is present, and a little exophthalmos may be present owing to the action of the superior oblique, which presses on the ball. If the lesion be in the oculo-motor nucleus, the near position of the nuclei of the fourth and sixth nerves will probably cause them to be affected also, thereby causing a general

FIG. 8.



Showing the nearness of origin of the oculo-motor (3), pathetic (4), and adducens (6). The roots of these nerves are shown by an incision which has divided the pons. iii. The third nerve, arising from several roots. iv. The fourth nerve. vi. The sixth nerve, arising from three roots. (Modified from Arnold.)

ophthalmoplegia (Fig. 8). If the lesion is not nuclear, it is due to disease in the nerve itself, as already pointed out. If this is the case, the lesion is probably due to pressure in the cavernous sinus or to periostitis of the bones forming the sphenoidal fissure through which the nerve passes (Plate I.). Sometimes, however, the paralysis of the nerve may be only partial, so that the external muscles of the eyeball escape, and only ptosis and a dilated pupil are present. Very rarely ptosis results from a cerebral hemorrhage, without the other signs of oculo-motor paralysis being present. That is to say, the branch of the oculo-motor which supplies the levator palpebræ is affected, while the branches supplying the external and internal ocular muscles escape.

If there is a history of a cerebral attack resembling a mild apoplexy, and a unilateral ptosis is present, the lesion is probably in the cortical centre for the oculo-motor nerve in the angular gyrus just below the inter-parietal fissure. The lesion is, of course, upon the side of the cortex opposite the ptosis. Such a case is very rare.

A fourth cause of ptosis is due to an affection of the sympathetic nerve, and is sometimes called pseudo-ptosis. There are associated symptoms of vascular dilatation, with redness and swelling of the skin of the affected side, elevation of temperature in that part, contraction of the pupil on the affected side, and apparent shrinkage of the eye into the orbit. This form of ptosis results from the paralysis of the unstriped muscular fibres of Müller which exist in the orbital fascia, for as these muscular fibres aid in holding open the lid their paralysis results in partial ptosis. Nothnagel asserts that such symptoms occur with lesions in the corpus striatum.

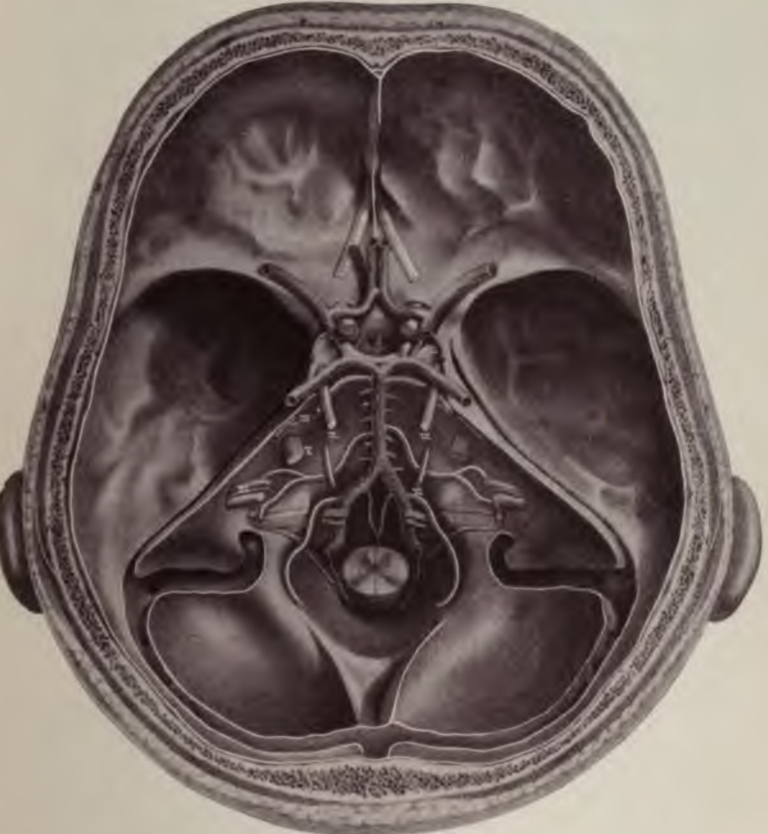
A fifth cause of ptosis is reflex irritation usually through the fifth nerve. This is probably due to an inhibition of the oculomotor centre. It is usually only transient.

Sixthly, it is not uncommon in cases of nervous syphilis for so-called alternate ptosis to develop. (Fig. 9.) First, one eye is affected by ptosis, and then the other just as the first begins to improve or recover.

Ptosis has been known to complicate tetanus, probably as the result of reflex irritation of the fifth nerve.

Ptosis, either unilateral or bilateral, may arise from hysteria and idiopathic muscular atrophy. If from hysteria, the diagnosis can be made from the age, sex, and history of the patient, from the presence of hysterical sensory changes described in the chapter on

PLATE I.



Base of Skull showing the Oculo-motor Nerves (III) Passing Through the Sphenoidal Foramen. (Modified from Arnold.)

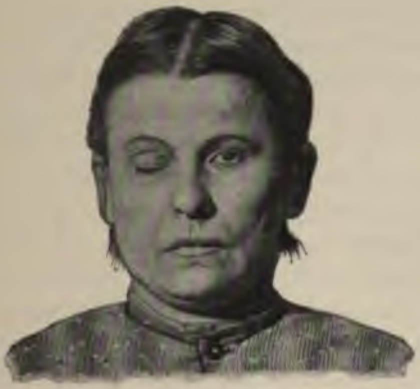
The other cranial nerves are also numbered and it is easy to see how an inflammatory exudate at the base might involve many of them.

III.—The oculo-motor nerves. IV.—The pathetic nerves. V.—The trifacial nerves. VI—Abducens. VII.—The facial nerves.

the skin, and from the fact that there is a tendency to spasm of the orbicularis muscle when the patient is made to look up. This contraction of the orbicularis proves that there is no true paralysis of the levators. If the ptosis is bilateral and hysterical, the head is tipped back when the patient is told to look up.

Single or double ptosis is by no means a rare symptom of locomotor ataxia, and is often associated with other evidences of oculomotor palsy. Sometimes diplopia due to this disease is the first symptom complained of, and the patient may state that the diplopia comes and goes.

FIG. 9.



Ptosis in a case of alternate hemiplegia of syphilitic origin. (DERCUM.)

Bilateral ptosis, like unilateral ptosis, may arise from tubercular or syphilitic changes about the base of the brain owing to pressure on the cranial nerves (see Plate I.), or it may be congenital, or if transient be caused by poisoning by gelsemium or conium. It is also seen in slight degree in feeble, overworked women, particularly in the early morning on awakening.

Again, it is not very rare to see slight drooping of both lids in all the members of a family, in which case the condition is usually most marked in the women, and is to some extent combated by the frontal muscles, which, in contracting, make the patient frown and draw up the eyebrows. (See chapter on the Eye.)

Ptosis, with hemiplegia of the face and limbs, on the opposite side of the body, associated it may be with hemianæsthesia, is due to a lesion in the crus cerebri, provided the two sets of paralysis occur simultaneously, otherwise they may be due to two separate lesions. (Hughlings Jackson.)

A very rare condition, of which there are but twenty-seven cases on record according to Darquire, is recurrent paralysis of the oculomotor nerve on one side. The attack begins with violent pain on one side of the head, nausea, and vomiting, and these symptoms are followed by ptosis, external strabismus, mydriasis, paralysis of accommodation, and crossed diplopia. It is seen most frequently in women, but may date from as early a period of life as eleven months. The attacks may last for a few weeks, and recur often or only after a lapse of many years.

As already stated, a form of alternate ptosis sometimes also develops in syphilitic persons.

Bilateral Facial Paralysis is a rare condition, and when it occurs can only be due to a bilateral lesion in the cerebrum, to acute bulbar paralysis, to progressive bulbar paralysis, to a lesion in the pons just where the facial fibres decussate, to bilateral disease of the pons owing to disease of the basilar artery, syphilis at the base of the brain producing a tumor or inflammatory thickening, very rarely to bilateral inflammation of the stylo-mastoid foramina, resulting from cold or double otitis, from toxic multiple neuritis, but not from that toxic neuritis due to alcohol. Very rarely bilateral facial paralysis results from multiple neuritis in its diphtheritic form.

The development of bilateral facial paralysis due to a double cerebral cortical lesion never occurs without evidences of paralysis elsewhere in the body, such as monoplegia or hemiplegia.

The bilateral paralysis of the facial nerve in acute bulbar paralysis is characterized by the limitation of the paralysis, as a rule, to the neighborhood of the lips, by dysphagia, lingual paralysis, affected speech, paralysis of the ocular muscles, and a rapid pulse. This disease is very rare, and depends for its existence upon an acute inflammation or myelitis of the medulla oblongata.

When due to progressive bulbar paralysis (glosso-labio-pharyngeal paralysis) the paralysis is confined chiefly to the lips, and is associated with alterations in the tongue (see chapter on the Tongue) and speech, with tremor of the tongue and stiffness of the lips. The mouth stands half-open, the lower lip is pendulous, and the patient's expression is that of a person about to burst into tears. The symptoms of glosso-labio-pharyngeal paralysis may, however, be exactly reproduced by diphtheritic paralysis, with this difference in prognosis: the first type always die and the second type usually get well.

In making a diagnosis of bulbar paralysis it should be remembered that another condition exists in rare instances in which no definite pathological changes can be found in the nuclei in the medulla oblongata, and yet many of the symptoms manifested by the patient are identical with those of glosso-labio-pharyngeal paralysis (true bulbar paralysis). This condition has been called "asthenic bulbar paralysis," and in it we find, as early symptoms, that the muscles of swallowing and of speech become easily tired on exertion, showing failure of the nuclei of the fifth nerve; that defects in articulation and speech are developed, indicating disorder of the nuclei of the ninth and tenth nerves; and clumsy movements of the tongue are present, which is a sign that the nuclei of the hypoglossal and twelfth pair are involved. These symptoms are practically identical with those of true bulbar paralysis. What are the symptoms which by their presence in the true disease and their absence in asthenic bulbar paralysis aid us in separating the two affections? The answer to this question is that the drooling of saliva, the atrophy of the tongue, lips, and extremities, the fibrillary twitching of the affected muscles, and the loss of electrical irritability in these muscles, all of which symptoms belong to true degenerative bulbar paralysis, are not to be found in the so-called asthenic form. There is, however, in the latter disease a condition rarely found in the degenerative form, namely, paralysis of the oculomotor, the lower facial, and the inferior division of the fifth or trifacial nerve, causing dilated pupils, diplopia (which, however, is not accompanied by strabismus), and ptosis (from the oculo-motor failure), facial paralysis about the mouth (from facial nerve failure), and loss of expression about the eyebrows and forehead (due to facial and trifacial failure.) Whether the diagnosis be true degenerative bulbar paralysis or the asthenic form just discussed, in both the prognosis is most unfavorable. Indeed, the asthenic form is often the more rapidly fatal of the two. In the latter the nuclei in the pons are probably always involved, but, as already stated, no pathological changes have been demonstrated in any of these nervous centres.

A very rare affection is oculo-facial paralysis, which is congenital or develops in childhood, and is chronic. There are present paralysis of the facial muscles and ptosis.

Facial Spasm. Spasm of the facial muscles may result from functional and organic disease, and occurs far more frequently in women than in men. The cause of the functional forms we do not understand, as they occur in neuropathic persons and about the climacteric period. Rarely the spasm arises from reflex irritation through the trifacial nerve, resulting from a decayed tooth or a cause in the eye or in the skin. Habit-spasm arises from an acquired trick. The organic causes are many. Thus there may be an irritative lesion of the facial nerve-trunk or one in the cortical centre for the face, a tumor pressing on the nerve at its point of origin, or an aneurism of the vertebral artery. The spasm may be confined to one side or distributed over both sides, and may be clonic or tonic in type. Sometimes it occurs only on attempted movement; in other cases it is constant. The clonic form is the more common.

Spasm of the face is seen in chorea, convulsive tic, blepharo-facial spasm, in tetanus, meningitis, and epilepsy. When due to chorea, it nearly always is clonic or twitching, as it is also in convulsive tic and habit-spasm, but in tetanus, meningitis, and epilepsy it is generally rigid or tonic. In chorea the spasm is most marked about the corner of the mouth and the eyebrow or eyelids. The movements of convulsive tic are exceedingly sudden, darting across the face and involving all the muscles supplied by the facial nerve. As a rule, this affection is unilateral. These spasmodic movements of convulsive tic may be almost constant or appear in paroxysms, and rarely the muscles of the jaw, the neck, and tongue are affected. The disease depends upon a disorder of the facial nerve, or its centres, which is not understood. The prognosis is bad so far as cure is concerned. Spasm of the levator palpebræ superioris muscle is sometimes seen as a symptom of exophthalmic goitre. It is called "Abadie's sign."

In blepharo-facial spasm there are paroxysmal spastic contractions of the orbicularis palpebrarum and other facial muscles. The spasm often tightly closes the lids. Generally in children there is also photophobia with the spasm of the eyelids, which is often tonic in character and generally bilateral. This condition has associated with it what have been called "Graefe's spots,"[1] namely, the presence of spots near the supraorbital foramen or over the vertebræ, which when pressed on cause sudden relaxation of the spasm. These should always be sought for, as they aid us in giving relief to the patient.

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[1] This term should not be confused with the more common term "Graefe's sign" used to indicate the condition in exophthalmic goitre in which the lids fail to follow the eyeballs when the patient looks down.

Spasmodic movements about the eyes such as have just been described are sometimes paralleled by what is called nictitating or clonic spasm, which is probably due to some undiscovered cause of reflex irritation.

The development of facial spasmodic twitching accompanied by a sudden burst of explosive speech, repeating the last word heard or said by the patient in conversation (called echolalia), or the sudden bursting out with some blasphemous or filthy word (called coprolalia), is sometimes seen in neurotic adults or children, and is often associated with perversion of moral sense. It is called by Gilles de la Tourette "Maladies des tic convulsifs," but this is an unfortunate term, because it is apt to be confused with ordinary convulsive tic of children or adults. (See Electric Chorea and Myoclonus Multiplex in the chapter on the Hands and Arms.)

In tetanus the muscles of the jaw, the masseters and temporals, are first involved in the tonic contractions, and these are followed by rigidity of the muscles of the neck and body. Often the *risus sardonicus* is marked from the first, and the face soon looks like that of a very old man owing to the muscular contractions.

In meningitis the characteristic symptoms which label the malady render facial spasm a comparatively unimportant symptom, and in epilepsy the convulsive seizure soon makes easy the diagnosis of the cause of the facial spasm unless the epilepsy is limited in its character, when the history of the presence of an aura, or of unconsciousness, or biting of the tongue may be discovered.

Spasm or contractures of the muscles of the face sometimes follow facial paralysis as recovery begins, and the contractures involve the formerly paralyzed muscles, whereas in paralysis in the limbs the contractures generally take place in the muscles which are not paralyzed. Sometimes these contractures in the face are permanent, and are due to incomplete restoration of the functions of the muscles affected. Care should be taken to remember that not very uncommonly contractures in the muscles of the face result from hysteria, and that they are often on the side opposite the facial paralysis if the latter exists.

Active spasm of the muscles of the face may follow exposure to cold, and it sometimes follows the paralysis due to this cause, or, in other words, is a sequence of Bell's palsy.

The Head and Neck.

In examining the head we look for variations from the normal in its shape, its fontanelles if the patient is a young child, the position in which it is held, and its movements as governed by the cervical muscles. Of the last we shall speak first, although they have been mentioned under the heading of wry-neck. We find that the head is moved abnormally in nodding spasm, in chorea, and in tetanus and strychnine-poisoning. It is also thrown backward and forward or from side to side in epilepsy, and in hysteria or in the convulsive seizures occurring in young children.

Nodding spasm of the head, depending upon somewhat rhythmical contractions of the sterno-mastoid and trapezius muscles, is sometimes seen in half-fed or rickety children. It also occurs in hysterical women, and in men who are not hysterical. The nodding may be slow and infrequent, only coming on with excitement, or it may be practically constant. It always becomes worse when the patient is examined, and may be so rapid and forcible as to seem almost severe enough to shake the head off the shoulders. Often the muscles involved will be found very rigid.

If the spasmodic movement be not rhythmical, as it usually is in nodding spasm, and yet be more or less constant though irregular, the cause is probably chorea minor if it is present in a child, or it may belong to the irregular movements of adults classed under the various forms of tic or choreiform spasm. (See chapters on Hands and Arms and on Convulsions and Spasms.)

Wry-neck consists in a drawing of the head to one side by spasm of the sterno-mastoid muscle, and at the same time the head may be tilted backward or forward according to the accessory muscles which may be involved in the spasm.

Sometimes a tonic spasm of the sterno-mastoid muscle, produced by exposure to cold or due to a distinct nervous lesion, causes the head to be drawn down over the shoulder, or bilateral spasm of this muscle causes fixation of the head. If the cause be exposure, with resulting myositis, the history of exposure, combined with that of a sudden onset, will permit a correct diagnosis and a favorable prognosis, it being remembered, however, if the patient is a female, that hysterical spasm may be the cause. If hysteria is the cause, the history of the patient, the presence of alteration in her color-fields and the other signs of hysteria can probably be elicited (see chapters on the Eye and on the Skin). On the other hand, if the contraction has come on gradually, after some injury or in association with some nervous affection elsewhere, it is probable that a true nervous lesion underlies the disorder.

If it is a tonic spasm, the involved muscle is on the side toward which the head is drawn, and the muscle on the opposite side is seen to be prominent owing to its being stretched by its opponent. The chin is, moreover, directed upward and away from the affected side. Rarely the trapezius is the only muscle involved, in which case the head is drawn backward and toward the diseased side, or, if the sterno-mastoid and trapezius muscles are both involved, the head is tilted laterally and backward until the patient looks up in the air. Pain in the muscles only occurs from fatigue. This tonic spasm affecting the muscles which support the head can be separated from that occurring in tetanus by the fact that in tetanus there is a general diffusion of the spasm to other muscles, although in that form of tetanus called "head or cephalic tetanus" the diagnosis is more difficult. This form of tetanus usually has with it the following diagnostic points: there is a history of infection, the character of the onset is sudden, there are trismus, difficult swallowing, respiratory disturbance, and facial paralysis with rare involvement of the ocular muscles. The spasm in cephalic tetanus is also often increased by movement or by the attempt to take food. Strychnine-poisoning is also to be thought of, but the limited character of the convulsion excludes that condition.

Should the muscles be affected by a clonic spasm, the head is jerked about instead of remaining fixed.

Retraction of the head in children is an indication in many cases of serious brain disease, and commonly arises from a basal meningitis, probably as the result of an effusion into the ventricles. It is to be remembered that some of these cases recover, though such a result is rare. Again, we should not forget that caries of the cervical vertebræ may cause this position, or that tender and enlarged glands in the neck may produce such a result. Sometimes, too, it occurs after falls without there being any other indication of meningeal irritation. Rarely in neurotic babies retraction of the head, as a temporary symptom, accompanies attacks of indigestion.

Similarly in adults suffering from cerebro-spinal fever the head is often held in a retracted posture.

The posture of the head may also aid us in diagnosis when no spasm of its governing muscles exists. Thus, chronic deafness in one ear may cause the patient to hold one side of his head further forward than the other, in order to catch the sounds he seeks with the good ear, and pronounced strabismus may cause a child to so carry its head as to improve its sight and avoid diplopia.

Persons suffering from great mental depression with a tendency to melancholia often sit for hours with the head bowed forward with the chin resting on the chest.

The changes from the normal in the shape of the head are to a certain extent considered in that part of this chapter dealing with the symmetry and appearance of the face, but there still remain to be discussed the changes in the shape of the head as a whole. These occur in acromegaly, osteitis deformans, and in hydrocephalus, microcephalus, rickets, idiocy, myxœdema, and cretinism.

The head of hydrocephalus is greatly enlarged above the level of the ears, and this causes the face, already having a tendency to faulty development, to look small and wizened. The eyes seem somewhat bulging, the orbital plates are oblique, and the back of the head is flattened. Sometimes in true hydrocephalus the fontanelle remains pulsating for a long period. Again, in true hydrocephalus choked disk is sometimes manifested quite early. (See Chvostek's and Trousseau's Signs.) In microcephalus, on the other hand, the head is small and often narrow. Technically, the term microcephalus is applied to idiots whose heads are less than seventeen inches in circumference. Nearly always the head of an idiot is abnormally formed. The cretinoid head is large, heavy, and massive.

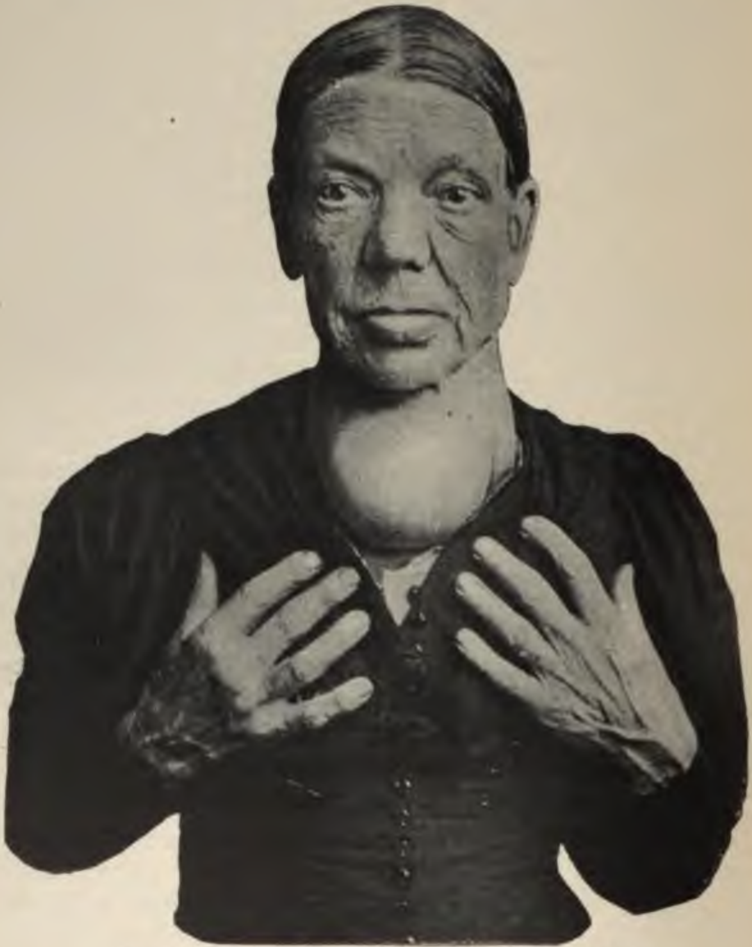
When a young child has unusually prominent parietal and frontal bones, which seem bulging, and there is a general resemblance in the shape of the skull to that of hydrocephalus, we suspect the presence of rickets. As a rule, the forehead is broad and high, the top of the head flat, and the shape of the head more round than in the genuine disease. Sometimes in such a child we find, in addition to these changes from the normal, spots of thinned bone in the occipital and parietal regions. These may be also somewhat softened, and this condition, called "cranio-tabes," is usually a sign of rickets which exists in association with infantile syphilis. Rickets is seen nearly twice as often in boys as in girls, and there is usually to be found deficient development of the bones everywhere, particularly in the ribs and legs. (For the rachitic rosary, see chapter on the Thorax.)

The condition of the fontanelles in young children is of importance in diagnosis. In the healthy child all the fontanelles save the anterior fontanelle close during the early weeks after birth, but the latter opening does not close entirely till the infant is about one year and a half old. During the first few months this fontanelle closes very slowly indeed, but after this time has elapsed its edges become rapidly approximated. The presence of other fontanelles in a child's skull after it is several months old indicates rickets, syphilis, hydrocephalus, or some intracranial growth producing pressure on the cranial bones, preventing their approximation. Generally, however, these minor fontanelles are not found open but closed, and the condition of the anterior fontanelle is the guide in diagnosis. In severe cases of rickets the anterior fontanelle remains open until the third or fourth year, and should the rachitic tendency be developed early in life the edges of the fontanelle may not only fail to be approximated, but may actually recede from each other. Sometimes if the edges of the fontanelle are found to be softer than usual the diagnosis of rickets can be so confirmed. If syphilis be the cause of the deficient bone development, evidences of this disease in mucous patches about the mouth and anus may be found or a history of heredity adduced; while if the condition be hydrocephalus the fontanelle will be markedly bulging.

If the skin over the fontanelle be found to be bulging temporarily to a slight extent, the cause probably lies in some acute disease with fever, producing cerebral congestion; whereas, if permanent, and if the general dimensions of the skull are not increased, an intracranial growth may be the cause, or a cerebral hemorrhage, a purulent meningitis, or some cystic formation may be present, or sometimes a thrombosis of a cerebral sinus produces hydrocephalus and bulging. In other cases thrombosis causes sinking-in of the fontanelle. This difference in the tension of skin over the fontanelle aids us in separating the meningeal symptoms of pneumonia from those of true meningitis, for in the true form the scalp is tense and in pneumonia it is often retracted.

Marked sinking-in or collapse of the fontanelle always indicates a grave condition arising from some disease which seriously weakens the heart and general circulatory system, particularly marasmus and cholera infantum. The other symptoms associated with this state

FIG. 10.

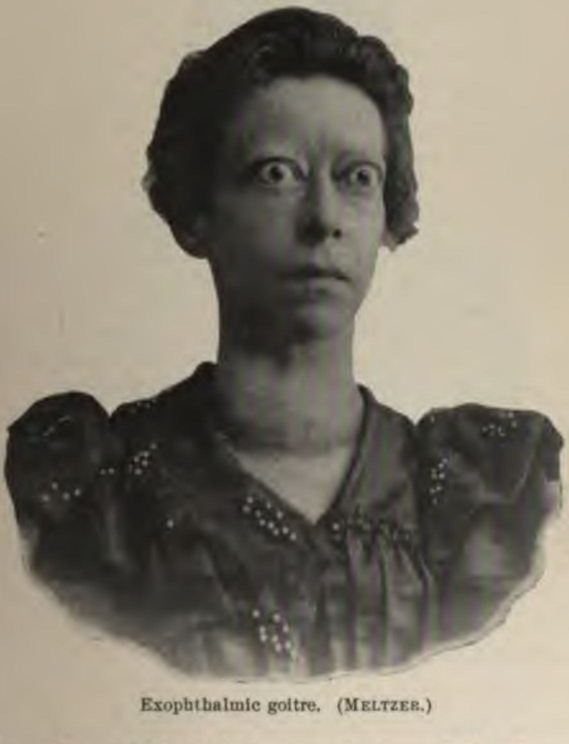


Acromegaly with goitre (not exophthalmic). (G. R. MURRAY.)

are usually a sunken appearance of the eyes, slight duskiness of the face, a cool skin, and a rapid feeble pulse. The patient is almost comatose, and there may be slight convulsive seizures. Such a condition has been called the "hydrocephaloid state," and has been confused with symptoms of cerebral effusion arising from tubercular meningitis. If there be marked diarrhœa present the following table of Symes will serve to clear the diagnois :

HYDROCEPHALOID STATE FROM DIARRHŒA.	CEREBRAL EFFUSION (AS IN TUBERCULAR MENINGITIS).
Diarrhœa.	Constipation.
No ocular paralysis.	Ocular paralysis and squint.
No rise of temperature.	Slight feverishness.
No headache.	Headache (if old enough to complain).
No tension or bulging of fontanelle.	Bulging fontanelle.
No rigidity and	Rigidity and retraction of head in many cases.
No retraction of head.	

FIG. 11.



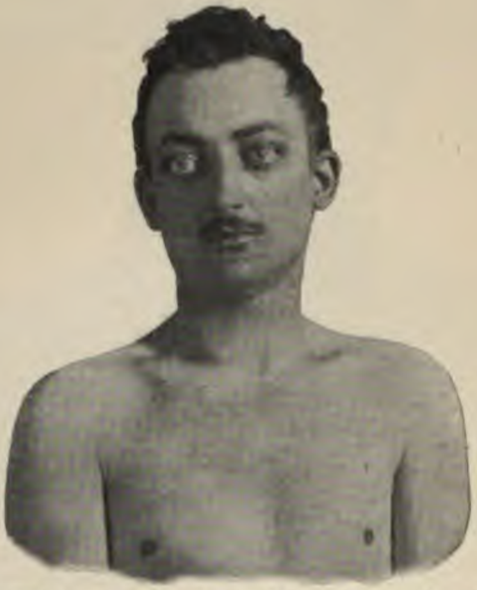
Exophthalmic goitre. (MELTZER.)

Sometimes in rachitic babies auscultation of the fontanelle will reveal a murmur, hæmic in origin. This is most frequently heard in this class of patients, but can occasionally be heard when no such disturbance of nutrition exists.

Excessive sweating of the head, producing a wet pillow, is often an indication of rickets when it occurs in a child.

A swelling in the neck in the median line, or on both sides of the median line, anteriorly, is probably due to goitre. (See Fig. 10.) If it is associated with cardiac palpitation and distress, exophthalmos, tremor, nervousness, and depression of spirits, it is called exophthalmic goitre. (See Figs. 11 and 12.) If these symptoms are absent, the condition is simply one of overgrowth of the thyroid gland.

FIG. 12.



Exophthalmic goitre in a male. The photograph does not clearly show the enlargement of the thyroid. (From a patient in the author's wards.)

Aside from swelling of the glands of the neck due to syphilis, Hodgkin's disease (see chapter on Fever), struma, and tuberculosis, there may be enlargement of the parotid gland on one or both sides, just in front of the ears and extending under the angle of the jaw. This swelling may be due to the specific inflammation involving these glands, known as mumps, or be due to other infections, such as typhoid, typhus, and pyæmic fever. If the latter be the cause, suppuration usually ensues. Rarely enlargement of the parotid glands follows trauma or disease of the abdominal viscera or pelvic organs. Sometimes the enlargement is chronic after the acute inflammation is passed by.

(For the movements of the head in epilepsy and hysteria, see the chapter on Convulsions.)

CHAPTER II.

THE HANDS AND ARMS.

The general appearance of the hands and arms—The shape of the hands in disease—Spasms of the fingers—Tremors of the hands—Paralysis of the hands and arms.

THE appearance of the hand and arm often gives us valuable hints in the diagnosis of disease, chiefly by reason of variation in their shape, manner of movement, and general consistency; but as all these conditions vary widely in normal individuals, we can only regard distinct and well-marked alterations from the normal type as indicative of a definite disease. We can, however, often gather general information as to the patient from the hands, particularly as to his occupation; thus we see the smooth, soft hand of the professional man or clerk, the horny hand of the laborer, the blackened nails and skin of the machinist, or the blue-black dottings of the hand of the miner; and Hirt asserts that atrophy of the antithenar eminence often ensues in cabinet-makers, perhaps from the excessive use of the plane. Even when no pathological condition exists we are wont to regard the heavy and somewhat thick and clumsy hand as an evidence of a phlegmatic temperament, and the thin, wiry, dexterous hand as indicative of the nervous temperament. Similarly, we recognize as the hand of the strumous that one in which the fingers are slender between the joints and the joints themselves thick and clumsy, or, again, in persons with tubercular tendencies, we see a slender, delicate hand, easily compressed and somewhat effeminate in type. Very commonly, too, in children who have developed heart disease in early life the hand becomes square-looking, and the fingers are club-shaped through thickening at the tips. A similar clubbing also manifests itself in many cases of emphysema and chronic phthisis in adults, and unilateral clubbing with incurvation of the nails of one hand is sometimes seen in thoracic aneurism.

From the appearance of the nails we can often gain important information; thus, whenever the color of the blood in the capillaries under the nails is dusky we know that a deficient pulmonary function exists or that the circulation is impaired, it may be from feebleness or from cold. In anæmia the nails are often very pale, and Stephen Mackenzie has asserted that if pressure on the tip of the finger completely empties the capillaries under the nails so that the appearance is pale the red corpuscles are present in only half the usual number.

White spots in the nail may be due to injury of the matrix by picking at the base of the nail, or be due to acute fevers producing trophic changes.

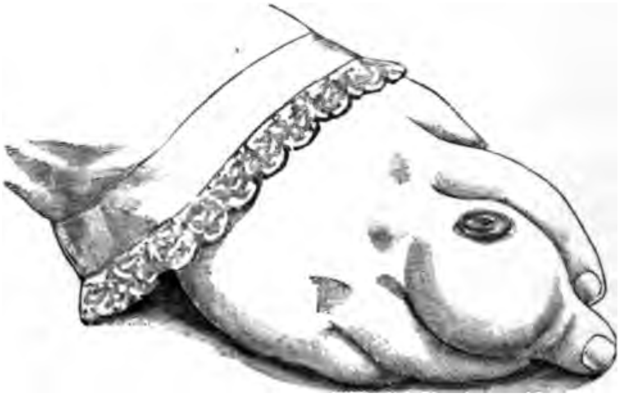
When the nails are striated and in longitudinal ridges the patient is often of the gouty diathesis, while transverse ridges may indicate arrest of nail-growth through local injury to the matrix or the impairment of the general nutrition as the result of some severe systemic shock, such as a severe surgical operation or prolonged illness. Sometimes these marks result from a severe attack of gout, and Fothergill tells us that it took about seven months for such a mark to grow out of his nails. Ordinarily, this mark will be found about half-way up the nail three months after the attack. In hemiplegia or acute infantile palsy the growth of the nail of the paralyzed part is generally arrested, as can be determined by staining it and watching it from day to day to see if the stained part gradually moves away from the base. When the nails are distorted and thickened the cause may be local injury or peripheral neuritis, or any condition of the nervous system resulting in decided trophic influences, as in that rare condition syringomyelia.

Hypertrophy of the nails so that they are abnormally elongated is usually associated with thickening and the development of great fragility. The nail may even be spirally twisted (onychogyrophosis), or, if very wide, may cut into the skin and produce paronychia. These conditions may result from skin lesions, such as eczema or lichen ruber, at or near the matrix, or be due to syphilis, and Vogl asserts that mere thickening may arise from severe fevers. They may also be seen in cases of Raynaud's disease, or in sclerodactyle, and in cases of pulmonary osteo-arthropathy.

Atrophy of the nails may apparently arise from causes identical with those which produce hypertrophy, and Kaposi has seen the nails soft and membrane-like, with abscesses under them, from psoriasis of the fingers.

A diagnostic indication given us by the fingers is seen in dactylitis due to syphilis (Fig. 13). Similar deformity, often multiple, also occurs in scrofulosis or tuberculosis. In other cases this is replaced by an eruption on the skin of the hand characteristic of syphilis. Another indication is seen in the ulcers at the bases of the finger-nails, with ecchymotic spots on the skin, produced by the chloral-habit; and still another is the sores seen at the bases of the finger-nails in persons who handle irritating drugs, such as elaterium. (See chapter on the Skin.)

FIG. 13.



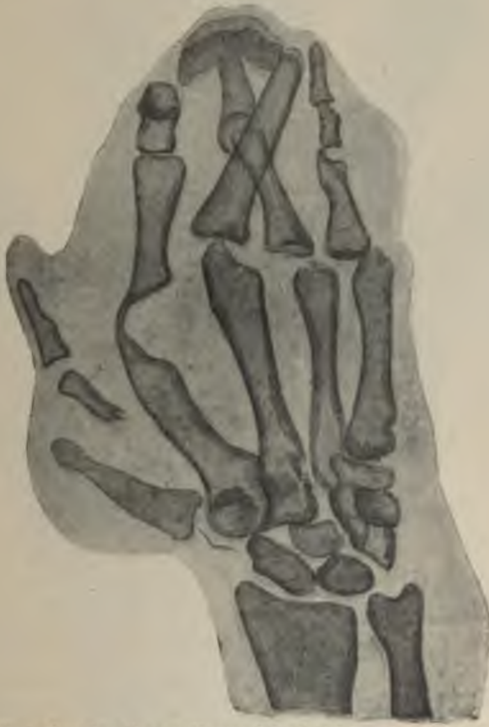
Dactylitis syphilitica in the infant. (TAYLOR.)

Congested veins on the hand may indicate obstruction to the venous circulation of the arm, or general lack of vascular tone and a feeble heart.

When the hand is cold and clammy the condition may be due to bromidrosis, or a local disturbance in innervation of the sweat-glands. It is often seen in cases of so-called spinal irritation and nervous exhaustion. Excessive sweating of the hand is also often seen in cases of progressive muscular atrophy.

There are two sets of movements associated with the muscles of the wrist and hand which possess grave prognostic and diagnostic importance in the presence of exhausting fevers. The first of these is twitching of the muscles of the forearm (subsultus tendinum). It indicates severe and dangerous disease. The second is picking at the bedclothes. The grave import of this dangerous symptom, "picking at the bedclothes," or carphologia, is recognized by Shakespeare in his description of the death of Falstaff: "After I saw him fumble with the sheets, and play with flowers, and smile upon his fingers' ends, I knew that there was but one way; for his nose was as sharp as a pen." And again, Hippocrates has well emphasized the gravity of this symptom, for he says: "In acute fevers, in peripneumonias, in pleuritis, and in headaches, the hands are moved to and fro about the face, seeking in the void, as if gathering bits of straw, picking at the coverings, or detaching objects from the walls of the room, constituting so many signs of a fatal termination."

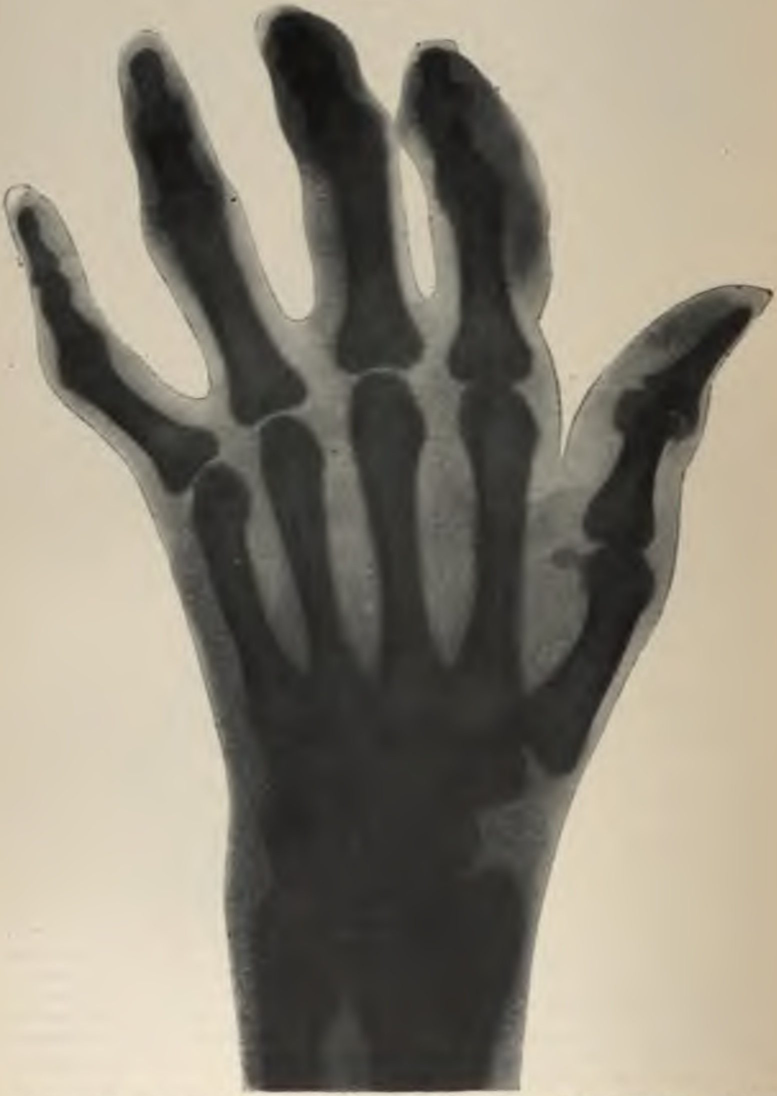
FIG. 14.



A Röntgen ray picture showing the condition of the hand in a case of chronic gout. It is seen that the tophi are comparatively transparent to the radiations. The changes to be seen in the bones are atrophy-erosion and dislocation; the thumb has been completely dislocated, the phalanges being altogether displaced; there has been anchylosis of the proximal phalanx of the first finger with its metacarpal, and a large erosion is shown on the ulnar side. Smaller erosions are shown on the metacarpal of the fifth finger at its bone, and also in the phalanges of the same finger. (From the *Medical Chronicle*.)

The fingers are often distorted and twisted out of their normal position from the trophic changes which take place in gout and arthritis deformans (rheumatoid arthritis). In gout the deformity invades the small joints in particular, and in many instances appears most marked in the forefinger. Fixation and deformity of the

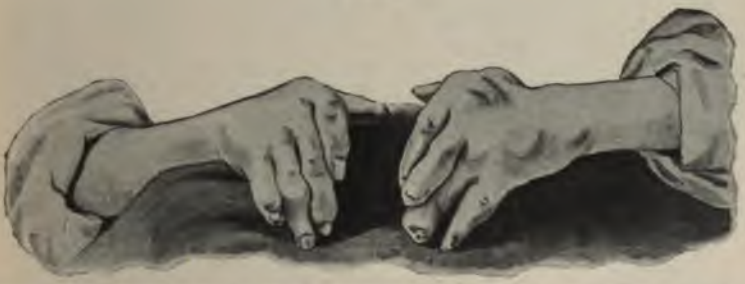
FIG. 15.



Radiograph of a case of gout in which the gouty deposits affected all the joints of both hands, greatly deforming them. The outline of the hand shows the deformity, but in this case it is interesting to note that the gouty deposits do not materially prevent the passage of the rays. Marked gouty nodules were present in this case, from which chalky material was readily obtained, and the outline of these can be readily discerned in the picture, particularly in the forefinger. (From the author's wards.)

fingers occur through the deposit of urate of sodium in large amounts about the joints in their tendons and sheaths, so that the fingers are as in splints. The knobs of urate of sodium appear as hard, white masses, and, if very superficial, as glistening masses, the surfaces of which often break down and allow the escape of material looking like powdered wet chalk. The joint-surfaces themselves are not primarily much altered, but secondarily grave changes occur in them. (Figs. 14, 15, and 16.)

FIG. 16.



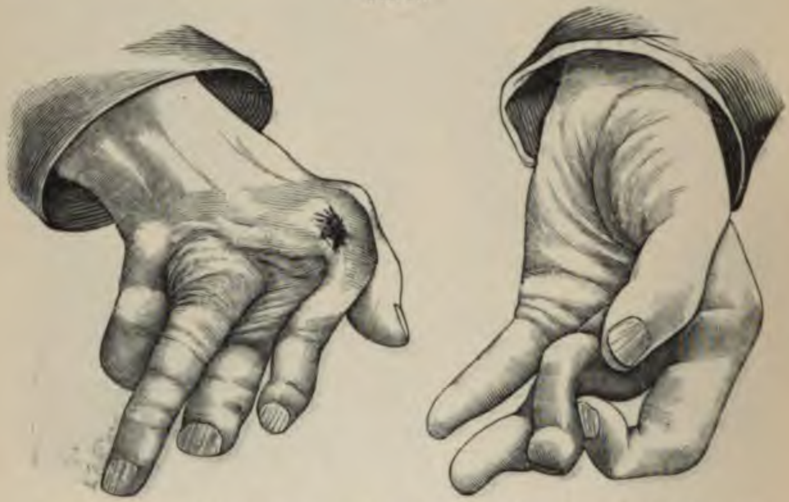
Same patient's hands as in Fig. 14, showing the appearance and explaining to some extent the superimposed position of the bones, but from this picture alone one would hardly expect such serious bone lesions. The hand to the left is a seal-fin hand.

Very commonly in gout the only joints of the hand which are involved are the first joints of the fingers, a knob developing on either side of the knuckle. (Fig. 19.) The little finger in gout is often bent at an acute angle at the middle knuckle, so that it is held in an awkward hooked position. (Fig. 17.) This is most commonly seen in women, while in men it is common to see forced flexion of the first phalanx of the middle finger into the palm of the hand, even when very little if any deposit of urates has taken place. This drawing down of the fingers is considered by Paget to be pathognomonic of gout, although the patient will claim that it is due to the use of a cane, a hammer, or other extraneous cause. (Fig. 18, hand to the left.)

Distortion of the hand with drawing of the finger or fingers into the palm may be due to Dupuytren's contraction, which results from burns or other injury to the palmar fascia.

While the history of the patient, the localization of the manifestations of the disease, and its character render a differential diagnosis between the hand of gout and that of arthritis deformans a possibility, it should not be forgotten that the deformities of gout may take every position assumed by those of arthritis deformans.

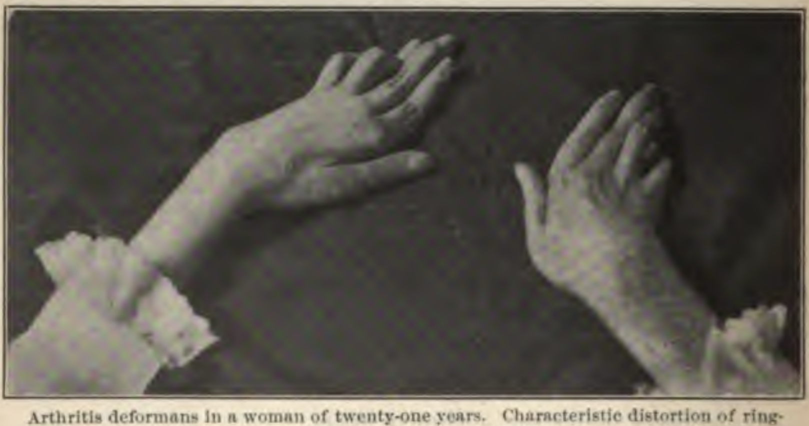
FIG. 17.



Tophaceous gout of hands, illustrating deflection and torsion of digits and phalanges. The figure to the left shows the "seal-fin" type. (DUCKWORTH.)

In arthritis deformans the distortion of the hand may be far more marked than in gout, for here there is not a splint-like deposit about the joint, but in its stead the development of exostoses on the edges of the articular surfaces, which at once lock and disjoint the fingers,

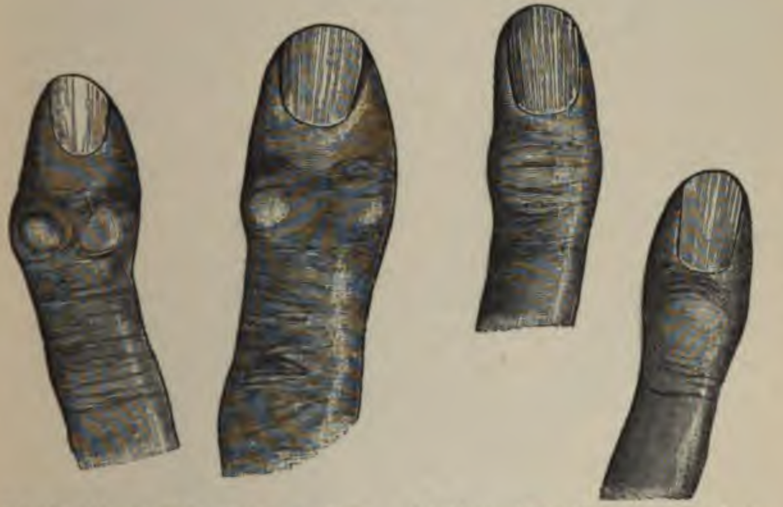
FIG. 18.



Arthritis deformans in a woman of twenty-one years. Characteristic distortion of ring-finger on right hand and seal-fin hand on right side. (From the author's wards.)

while at the same time the opposite side of the joint may be partially absorbed, so that dislocation is still more readily produced. (Fig. 20.) As a result there is sometimes developed what is called

FIG. 19.



Heberden's gouty nodes. Illustrating common forms of terminal phalangeal deflection. Forefinger and little finger of a woman aged seventy years. "Crab's-eye" cysts over the joints are also depicted. (DUCKWORTH.)

Nodular swellings (Heberden's nodes) due to gouty arthritis on the forefinger and little finger of a woman aged fifty years. (DUCKWORTH.)

the "seal-fin hand" (also seen in cases of gout), a hand in which the digits are deflected chiefly toward the ulna, through the action of the extensor muscles, which are supplied with nerves which are reflexly irritated by the condition of the joints, and thereby cause spasm (Charcot). (Figs. 16, 17, and 18.)

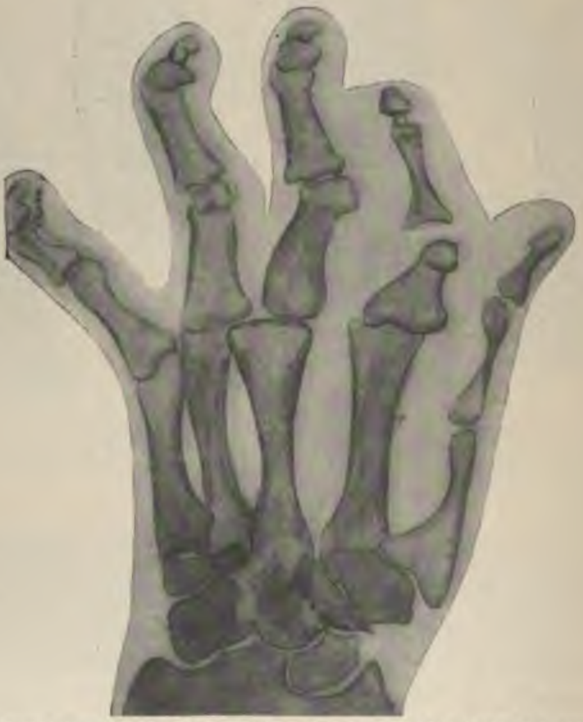
The term Heberden's nodes is applied by some to the exostoses at the sides of the phalangeal joints met with in rheumatoid arthritis or arthritis deformans; by others, as by Duckworth, to somewhat similar gouty deposits. These are sometimes called "Haygarth's nodosities."

Chronic rheumatism may produce gradual changes in the shape of the hand chiefly through disuse and the alterations which it causes in the capsules and ligaments. The chief alteration is immobility or stiffness. Some persons believe that when the hand wastes it does so not from disuse, but through reflex nervous influences.

Chronic rheumatism rarely, if ever, occurs in the hands alone, but when it does the joints are often swollen and somewhat tender, but never hard as in gout.

The finger-joints are not commonly involved in acute articular rheumatism, certainly very rarely as the only manifestation of the disease. The inflammatory process is more apt to be about the ball of the thumb, or in the wrist and carpal joint. The hand is seen under these circumstances as a clumsy, swollen mass, puffy, and exquisitely tender and hot. Sometimes it is quite red at the joints, but otherwise quite pallid, particularly in the puffy, œdematous area on the back of the hand. The presence of intense local inflammation, the history of sudden onset, and the intense pain on movement

FIG. 20.



A Röntgen ray picture showing the condition of the bones of the hand in a case of chronic rheumatoid arthritis. It will be seen that the peculiar outlines of the proximal phalanges are due to their positions; as lesions we may note anchylosis of the metacarpal bone of the middle and ring fingers with the os magnum and unciform bones, with deposits in the heads of the phalanges and dislocations due to absorption. (From the *Medical Chronicle.*)

readily separate acute rheumatism from chronic gout and arthritis deformans, and leave it to be separated from sprain, septic arthritis, and deep-seated inflammation of the hand proper. The first is excluded by the history, the second by the history and general lack of evidence of gonorrhœa or sepsis or purpura, and the third by the lack of accompanying general systemic disturbance and the absence of a history of traumatism or infection. In this connection it should not be forgotten that synovitis of the joints of the hands, wrists, and elbows sometimes occurs during the fall of temperature in scarlet fever, and is often not associated with any rise of temperature as a result of its development. The condition is sudden in onset and usually rapid in its course. The same state may exist in the joints of the lower limbs, but Marsden found it in the hands and wrists in seventy-two instances out of one hundred cases, and only twenty-five times in the larger joints out of a hundred cases. The condition usually appears, however, in rheumatic children and those with a rheumatic heredity, and is generally relieved by salicylates, so it is not a pure septic arthritis.

The nervous disturbances which change the appearance of the hands are very numerous.

Angioneurotic œdema is not peculiar to the hand, although frequently involving this part of the body. It consists of a swelling varying in size from a dime to a silver dollar, which is not œdematous in the sense that it can be pitted on pressure. This swelling, which may be multiple, red in color, or pale and waxy in appearance, lasts but a few hours or days, disappears, and often speedily returns. Somewhat allied to angioneurotic œdema is that condition of the hand (or toes) characterized by a white and waxy or slate color of the fingers, associated with coldness, swelling, and mottling of the skin, termed "Raynaud's disease." Often this is a passing condition, but in its severe forms there is finally developed dry gangrene in the fingers involved. The conditions of the hand resembling it, from which it must be separated, are senile gangrene, in which the advanced age of the patient and the presence of diseased and thickened bloodvessels will enable us to decide on the latter as the cause; frost-bite, in which the history of exposure will be of value, although exposure to cold often precipitates an attack of Raynaud's disease; ergotism, which can be discovered by the history of the patient having for a long time taken food which may have contained bad rye; leprosy, which will probably be seen more marked in other parts, and in the patches of which can be found the leprous bacillus; and alcoholic neuritis, of which we shall speak later (see chapter on the Skin). In that state known as Morvan's disease, or "pain-anæsthesia with whitlow," there is a slowly progressive loss of power in the hand, with atrophy and ulcers about the bases of the nails. Sometimes the terminal phalanges undergo necrosis, and enlargement of the fingers, through swelling, may be very marked. It is probable that this condition represents two separate lesions, namely, neuritis and syringomyelia, and it is an exceedingly rare disease.

Swelling of the hand, followed in some months by rupture of the skin, may, in a person from the tropics, mean mycetoma, which is, however, seen more commonly in the lower extremity as "Madura foot."

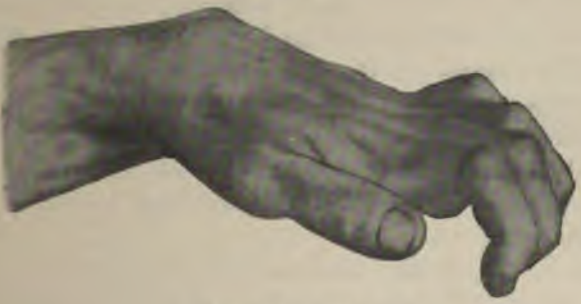
In addition to these trophic changes in the hand we have the so-called "spade-like" hand seen in myxœdema, acromegaly, and the pulmonary osteo-arthropathy of Marie. In myxœdema the deformity depends upon the alterations in the subcutaneous tissues, rather than on changes in the bones, so that the hand is swollen or boggy-looking, but does not pit on pressure as in true œdema. In acromegaly the enlargement is chiefly osseous, as it is also in pulmonary osteo-arthropathy, the formation being on a gigantic scale. In the latter disease, however, the hands and feet are alone affected, and the enlargement is not symmetrical. Further, this condition is nearly always associated with changes in the lungs, such as emphysema, tumors, and old bronchial troubles. The hands are not only greatly enlarged, but deformed, so that a side-view of the finger-tips reminds one of the shape of a parrot's beak, the nail being turned over the end of the finger. This is particularly well marked in the thumb.

The differentiation of pulmonary osteo-arthropathy from acromegaly is to be found in the fact that in the first-named disease there are no changes in the face, the skin, lips, or orbital ridges. Neither is there spinal kyphosis in the cervical region, although it may be present lower down. Again, in pulmonary osteo-arthropathy the long bones of the upper extremities are greatly enlarged in their epiphyses, while in acromegaly they are not so locally enlarged.

Alterations in the contour of the hand are, however, far more frequently produced by atrophic processes than by those which result in hypertrophy. They arise in cases of paralysis not only from wasting of the muscular tissues, so that hollows or sunken places occur, but also from the distortions caused by the contractions of healthy muscles, which, having no opposition as in health, speedily draw the bones of the hand into abnormal positions. In other cases the diseased muscular fibres may be spasmodically contracted, overcoming the resistance of the healthy muscles.

The wasting of the hand seen in old age, particularly in women, and in advanced phthisis, diabetes mellitus, and other conditions in which the tissues of the body in general lose their plumpness, is so universally distributed that a diagnosis of wasting from old age is not difficult. On the other hand, the wasting due to nervous lesions is generally not universal, but limited to single muscles or groups of muscles, the remaining portion of the hand having its normal appearance or being only indirectly influenced.

FIG. 21.



Claw-hand. (GRAY.)

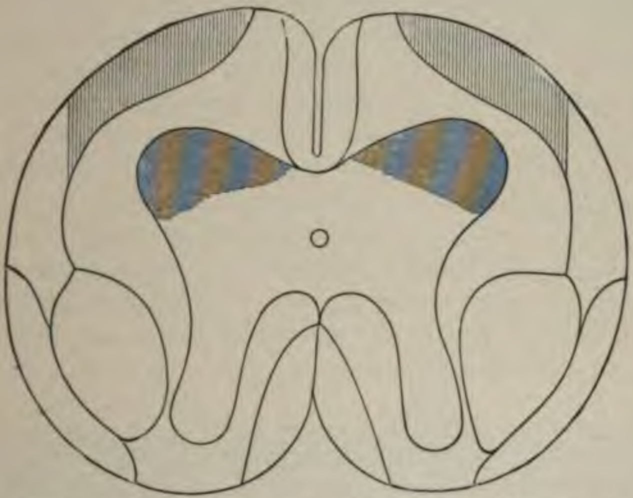
Under the name of "claw-hand," or "*main-en-griffe*," we find a deformity of the hand which is in itself very characteristic, although indicative of several causes which all operate in an identical manner. The back of the hand loses its normal convexity and becomes somewhat concave, the tendons on the extensor surface stand out in ridges, the proximal phalanges are drawn backward toward the wrist, while the second and third phalanges are drawn toward the palm of the hand (Fig. 21). Sometimes, however, the tips of the fingers are drawn toward the back of the hand. This deformity results from atrophy and paralysis of the interossei muscles and lumbricales, which are supplied by the median and ulnar nerves. The extensor communis digitorum and flexor digitorum produce a dorsal flexion of the first phalanges and a complete palmar flexion of the second and third phalanges. A certain amount of immobility is also caused by the fact that flexion of the hand is impossible in the fingers and almost lost at the wrist.

The claw-hand having been recognized, it remains to be decided what are its causes. It may be due to disease of the peripheral nerves (the ulnar and median), of the cells in the spinal cord, and of the cells in the cerebral cortex in the hand-area.

Taking up for consideration paralysis of the median and ulnar nerves as a cause of claw-hand, we find that the most common cause is a neuritis produced by some mechanical injury resulting from an accident, or from the following of some occupation in which, for example, the artisan presses his elbow constantly on some hard surface. The deformity may be, therefore, either unilateral or bilateral (generally the former), and there will be evidences of local injury, or a history which will indicate that the lesion is peripheral. Further than this, there will nearly always be found, in ulnar and median injury, sensory as well as motor paralysis; and Hirt asserts the remarkable fact that the claw-hand may develop in cases in which sensory disturbances are the only evidence of median and ulnar difficulty—in other words, before motility is lost through paralysis. (See chapter on the Skin, Anæsthesia of the Skin.) Toxic neuritis very rarely, if ever, causes claw-hand, as the musculo-spiral nerve is more commonly affected in this condition and the extensors become paralyzed.

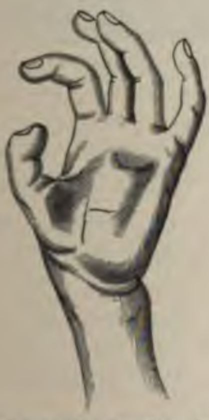
There are several spinal causes of claw-hand, the most important of them being progressive muscular atrophy, that disease in which there are atrophy and abnormal change in the anterior horns of the gray matter of the spinal cord, particularly in the cervical region. (Fig. 22.) It will be remembered, too, that the anterior nerve-roots and motor nerves become involved in this process. As a result of these changes, we have developed loss of power in the hand and arm followed by the development of a claw-hand from wasting of the same muscles, as already described, the disease-process being generally bilateral, but affecting the right hand and arm more than the left, as a rule. As progressive muscular atrophy often makes its first manifestation in these muscles, the hand affords much diagnostic information in suspected cases, and if the patient with this disease be watched as he unbuttons his coat, it will be found that he does not use his thumb and first finger, but pushes the buttons or the edge of the buttonholes with the back of his fingers. The additional symptoms are some pain or paræsthesia in the affected parts prior to the wasting, and the spread of the paralysis, as its name indicates, from muscle to muscle. (Figs. 23, 24, and 25.) Thus, beginning in the ball of the thumb it passes to the

FIG. 22.



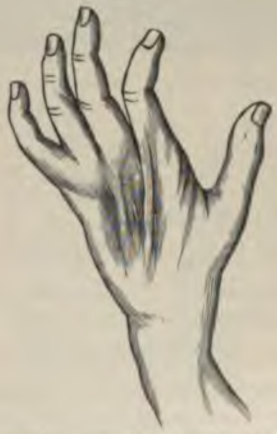
Areas of spinal cord involved in progressive muscular atrophy. The areas involved are the anterior horns of the gray matter chiefly (shading heavy) and the anterior lateral tracts and anterior root zones (shading light).

FIG. 23.



Progressive muscular atrophy. Ape-hand. (EICHHORST.)

FIG. 24.

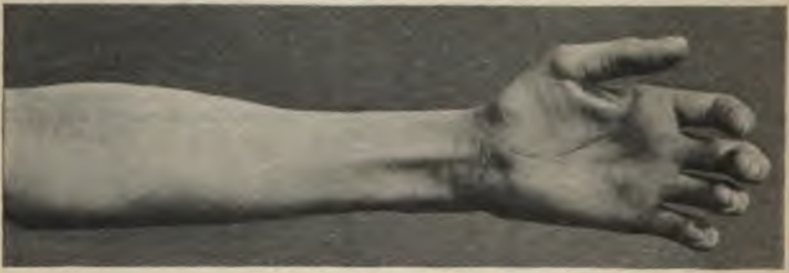


Progressive muscular atrophy. Sunken-in interosseal spaces on the back of the hand. (EICHHORST.)

interossei, and thence up the forearm and arm. Sometimes, however, the forearm muscles escape, and the shoulder muscles are attacked secondarily. Very rarely are the shoulder muscles first affected. Soon after this the dorsal muscles fail and lordosis begins, or the head falls forward on the chest. Finally, the respiratory muscles are attacked. The irritability of the muscles is increased, so that they contract if tapped, and fibrillary tremors constantly affect them in many instances. No vasomotor changes take place in the affected part, but, finally, the reactions of degeneration develop. The disease may last for many years.

Sometimes in chronic poliomyelitis in the adult a deformity somewhat like that of claw-hand may exist, but this is a very rare condition, comparatively speaking, and is separated with difficulty from the claw-hand of peripheral neuritis of a general and severe type.

FIG. 25.



Hand and forearm in chronic spinal muscular atrophy, showing especially wasting of thenar and hypothenar eminences. (DERCUM.)

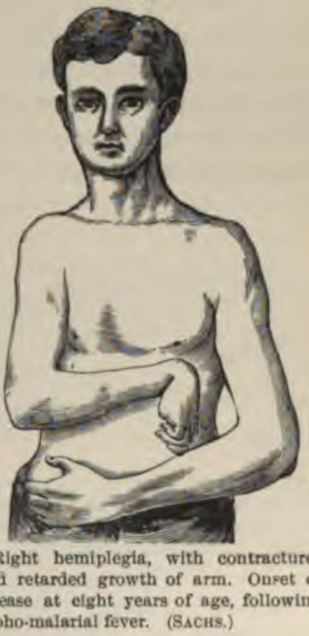
As the result of the acute poliomyelitis of infancy, we may also have the hand distorted by contractures, such as forced extension in paralysis of the flexors, forced flexion in paralysis of the extensors, and claw-hand in paralysis of the interossei, but in most cases of this disease the foot is the part involved in the disorder. In progressive muscular atrophy the atrophy often precedes the paralysis, whereas in poliomyelitis the paralysis precedes the atrophy, so that in the former the reaction of degeneration develops late, and in the latter develops early. A somewhat claw-shaped hand is also sometimes seen in that very rare condition called Morvan's disease, but it has not the characteristic appearance of *main-en-griffe*, there being a slow symmetrical wasting of the muscles with a drawing of the fingers into flexion. There are also analgesia and painless whitlows. It usually occurs in young or middle-aged males. Morvan's disease of the fingers, as already stated, may arise from a syringomyelia and neuritis, or neuritis alone.

Another spinal lesion producing great alterations in the appearance of the hand and arm, through wasting of the thenar and antithenar and interossei and the muscles of the arm, is amyotrophic lateral sclerosis. Here again the hand often shows the first manifestations of the disease in the loss of power of which the patient complains. The early symptoms of amyotrophic lateral sclerosis may closely resemble those of progressive muscular atrophy in the loss of power in the thumb muscles, but in this disease the reflexes are markedly increased in the affected muscles, whereas in progressive muscular atrophy they are lost, although fibrillary muscular twitchings may be caused by tapping. Again, the patient is usually manifesting some of the symptoms of lateral sclerosis when he comes before the physician, such as weariness, stiffness, and loss of power in the legs. (See chapter on Legs, Paraplegia.) There are also exaggerated knee-jerks and ankle-clonus, and wrist-jerk is marked.

Wasting of the muscles of the hand, causing distortion, may also be due to syringomyelia, but generally there will be, with this, loss of power and disturbance of sensation, such as anæsthesia. Often in syringomyelia there will be developed an arthropathy of the arms such as is seen in the legs in tabes.

Wasting of the hand, with flexion and rigidity and sometimes contractures, is seen rarely in advanced paralysis agitans in place of the characteristic tremor.

FIG. 26.

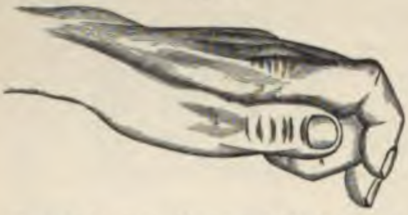


Right hemiplegia, with contractures and retarded growth of arm. Onset of disease at eight years of age, following typho-malarial fever. (SACHS.)

In the "cerebral palsy of children," sometimes called "spastic infantile hemiplegia," the hand may be flexed on the forearm, and the forearm on the arm, the thumb drawn into the palm of the hand and the fingers flexed as in Fig. 26. These deformities are not necessarily confined to one arm alone, but are sometimes bilateral. A peculiarity of these cases is that the muscles waste very slightly, and do not develop the reactions of degeneration, so that the case separates itself from poliomyelitis. The fingers in the cerebral palsy of children can often be placed in curious positions with ease, and, if the limb be suddenly flexed, a lock-like sensation will be imparted to the physician's hand. Convulsive seizures of an epileptiform type are very frequent in cases of cerebral palsy in children. Cohn asserts that there are on record eight cases in which intention-tremor has taken the place of the spastic rigidity just described, and he reports a ninth. Similar lesions may follow infantile cerebral hemorrhage, thrombosis, or embolism.

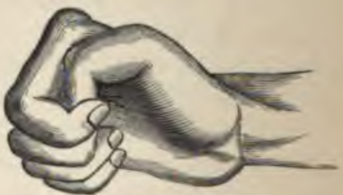
Again, in persons who have had apoplexy it is not uncommon as time goes on for the temporary spasm seen in the muscles of the hand and arm to be replaced by permanent contractions resulting in deformity. These contractions, if they occur early, are an evidence of irritation of the pyramidal tract or the fibres just behind the knee of the internal capsule, and are of serious import, as they indicate the extension of marked inflammatory processes. When they come on later they show that a degenerative process is descending the pyramidal tracts. Wasting finally comes on. (For further discussion of the significance of paralysis in the arm and hand, see succeeding pages and chapter on Hemiplegia.)

FIG. 27.



Hysterical atrophy of the hand with flexion of the last two phalanges into the palm, particularly the last phalanges of the index and middle fingers. (GILLES DE LA TOURETTE.)

FIG. 28.



Same hand in another view.

A very important point always to be remembered in examining contractures of the hand and arm, or of the lower limbs, is the fact that they often are due to hysteria, in which case the history is that they set in suddenly, and they are generally accompanied by other hysterical manifestations, which can be discovered if sought for.

As a rule, the muscles do not waste or develop degenerative reactions, but rarely such wasting may occur. (Figs. 27 and 28.) Care must be taken in giving a prognosis for cases of hysterical contracture, since organic lesions sometimes supervene. Charcot states that if the contractures persist when the patient is under anæsthesia, and the muscles are atrophied, organic disease exists. It is important to remember this, for these contractions may be practically permanent when once induced, and, as injuries may produce either a true organic or a false hysterical contracture, much medico-legal interest centres about this differential diagnosis. Closely allied to these cases are those of hysterical contracture, in which after grasping an object the patient cannot let go until the muscles are stroked. Putting an Esmarch bandage on such a forearm will usually produce the spasm.

When in the course of an acute illness in a child the fingers are drawn down into the palm of the hand, with the tips touching the palm and the thumb turned in beneath them, with its tip pressing the palm, the patient may have meningeal congestion or inflammation, or hydrocephalus, and a general convulsion may be imminent.

When the fingers are bent toward the palm, but the tips extended and the thumb turned in ("the accoucheur's hand"), the position is typical of tetany, but in this condition the rest of the body will give evidence of involvement. The nervous irritability in this condition is greatly increased, and pressure on a large bloodvessel or nerve-trunk will often produce the spasm. Curiously enough, gastric dilatation or thyroid wasting will often be found with tetany. In other cases it appears to be due to profound debility, as after prolonged nursing. (See Tetany in chapter on Convulsions and General Spasms.) Care must be taken to separate the so-called carpopedal spasm of rickety, hydrocephaloid children from true tetany, in which the body is usually involved, and from spastic paralysis due to infantile cerebral palsy.

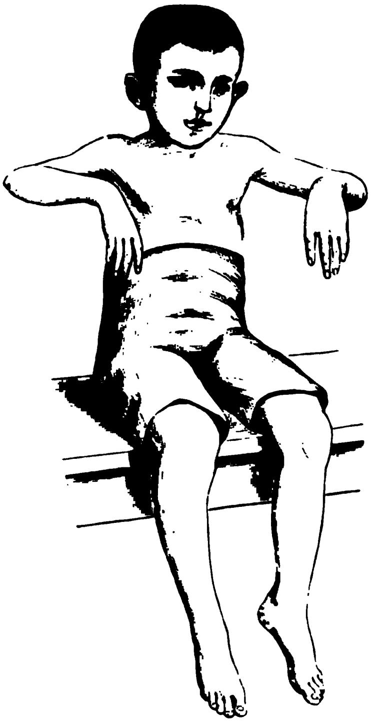
Spastic rigidity of the arms is often one of the earliest signs of chronic hydrocephalus, even before the skull begins to enlarge, and convulsions may be present from time to time. In congenital spastic rigidity due to sclerosis or defective development of the cortex cerebri the spastic condition is usually confined to the legs. (See chapter on Legs and Feet.)

Spasm of the fingers of a rigid type on attempting to make certain movements is also seen as the result of excessive use of the part involved, and occurs in seamstresses, cigarette-rollers, cigar-rollers, typewriters (rarely), telegraphers, milkers (rarely), persons who use a pen to excess, and in piano, flute, clarionet, and violin players, or in persons engaged in any occupation requiring constant and comparatively minute and well co-ordinated effort. It seems to be more common in men than in women by a large proportion (39 to 4).

Sometimes paralysis, tremor, or vasomotor disturbances take the place of occupation-spasm.

The spasm resulting from occupation must be separated from that sometimes seen in the hand in post-hemiplegic chorea, progressive muscular atrophy, the various forms of toxic peripheral neuritis, and that due to irritative cerebral foci, such as tumors of the brain. The history nearly always clears up the diagnosis. Spasm of the muscles of the hand and arm, rhythmical or otherwise, may also be due to hysteria, and may resemble, when due to this cause, true tetany (not tetanus).

FIG. 29.



Boy with multiple neuritis, with double wrist-drop and slight foot-drop. (SACHS.)

The position of the hand may be very various. Thus, the hand may drop edgewise from the radius toward the ulna in cases of rheumatoid arthritis (see Fig. 18), from paralysis of the extensors on the radial side of the forearm, resulting from neuritis or acute infantile poliomyelitis, while marked wrist-drop may occur from paralysis of the extensors in chronic lead-poisoning, or in any form of neuritis, toxic or otherwise, involving the nerve supply of these muscles (musculo-spiral nerve). (Fig. 29.) Wrist-drop may also be developed by pressure upon the musculo-spiral nerve, as in crutch-palsy.

If the wrist-drop is bilateral, it may be due to toxic neuritis; but if unilateral, it is probably, but not positively, due to pressure-paralysis from sleeping with the head resting on that arm, or from pressure by a crutch, or from some similar pressure capable of injuring the nerve. Very rarely unilateral wrist-drop is seen in lead-poisoning. When lead is the cause, the supinator longus usually escapes, as does also the short extensor of the thumb, so that the forearm can be flexed and the thumb extended. Pain is rarely present in pressure or lead wrist-drop, but is present in wrist-drop due to alcoholic and other forms of toxic neuritis. Often, too, in these cases the flexors are considerably involved (see part of this chapter on Brachial Monoplegia).

Choreic movements of the hands and arms in children are seen chiefly as a manifestation of chorea minor. They are usually met with in rheumatic and neurotic children, and heart-murmurs are generally to be heard in these cases. The first evidences of spasm may be developed in the hand, and be limited to that member in rare cases, and the hand often drops things that are placed in it. The hand itself is rarely involved alone, and the muscles of the arm toss the entire arm and hand with a fidgety, jerking movement which is very characteristic. A form of chorea minor, usually limited to the arm, is called paralytic chorea. It comes on suddenly, and is characterized by loss of power with a few feeble twitches. It affects only children. The same term, "paralytic chorea," is also applied to a condition sometimes seen after an apoplectic stroke, choreic movements taking place as degenerative changes in the muscles are developed.

Sometimes choreic movements come on in the latter half of life, often preceded by emotional disturbances. These movements are not true chorea. They are often called senile chorea.

In some cases of adult chorea the patient tends to become maniacal, particularly toward night. Such cases usually occur in women, and the prognosis as to life is bad. There is often in these cases great mental hebetude.

Several other affections which somewhat resemble true chorea are sometimes met with, but all of them lack, with one exception, the peculiarity of its movements. One of these is what has been called habit-chorea, or, more correctly, habit-spasm, in which condition the patient acquires a nervous trick of jerking a muscle or a set of muscles. Unlike true chorea, it is more frequently seen in adults than children. Its limitation, as a rule, to a single set of muscles and the history of the case usually separate it from chorea minor, and it is to be recalled that the movements consist in sudden twitchings rather than jerking, irregular muscular movements.

In paramyoclonus multiplex the disease, as the name implies, usually involves symmetrical parts, the contractions of the muscles appear in paroxysms, and the muscles involved are usually the biceps, deltoid, and triceps in the arms, and the quadriceps femoris and calf muscles of the lower limbs. Myoclonus multiplex is a disease of adult life, and chorea is usually seen in childhood. Sometimes the muscles in myoclonus are exceedingly irritable.

Under the name of electric chorea, or "Dubini's disease," Dubini described a disease, affecting both sexes and all ages, in which sudden shock-like contractions of the muscles take place, as if they were being stimulated by a slowly interrupted faradic current. The disease usually begins in the upper extremities, and gradually involves the rest of the body, and progressively passes to a fatal issue. This is a very rare disease, and the sudden contraction of the muscles in tonic spasm separates it from chorea.

Still another form of electric chorea is that of Bergeron, which is probably identical with what has been called hysterical chorea. Here, again, the shock-like muscular contractions are manifested chiefly about the shoulders. The patient is usually a female, and has the stigmata, sensory and otherwise, of hysteria. (See chapters on Skin, Eye, and Feet and Legs.)

Again, the physician may meet, exceedingly rarely (almost never in the United States or England), with a condition called convulsive tic or palmus, which has also been called "the jumpers," in which the movements are not in the slightest degree like true chorea, but are sudden muscular movements, usually imitative of the act of some other person or animal. This is often associated with echolalia —that is, repeated or echoed speech—or coprolalia or filthy speech.

Finally, another very rare disease is that known as Huntingdon's or hereditary chorea, a condition in which the twitching usually begins in the face and extends to the arms and legs. This ailment is hereditary, rarely begins before thirty years of age, is accompanied by progressive mental deterioration, a tendency to melancholia, and may last ten or twenty years.

Mercurial poisoning producing tremor may cause so coarse a movement in advanced cases that the case may be thought choreic. (For a description of tremors, see latter part of this chapter.)

In "Thomsen's disease" the hand is placed in tonic spasm as soon as voluntary movement is attempted. Closely resembling Thomsen's disease, or myotonia congenita, is what is called paramyotonia congenita, which exists in three forms: first, a patient suffering from paralysis agitans on attempting to move is seized with rigidity of the muscles, which holds him fixed; second, a patient is suffering from ataxia and muscular weakness, and is seized with an attack of muscular rigidity; and, third, a patient may have the muscular fixation occurring just as it does in Thomsen's disease, save that it is produced by cold or exposure, and not by intention-movement, and may last for hours. (See also Athetosis.)

Tremors of the Hand and Arm. The movements of the hand should always be carefully watched in cases of suspected nervous disease. The most common alteration from the normal will be found to be tremor, which may indicate paralysis agitans, disseminated sclerosis, general paresis, chronic mercurial, plumbic, or alcoholic poisoning, hysteria, senility, and Graves's disease. Sometimes a tremor may be found in naturally nervous women who are drinkers of tea to excess.

In paralysis agitans the whole hand is involved, and generally both hands are equally affected. The tremor is rhythmical and fine or minute in character at first, but later may be quite coarse. It is a slow tremor of about five vibrations per second, which is more or less constant, and worse when attention is called to it, but it is not greatly increased, and, perhaps, is even decreased, by a voluntary act, such as an attempt to raise a glass of water. Very rarely, however, the reverse holds true, and the tremor is increased by voluntary effort. The fingers are generally semi-extended and the thumb is adducted, so that it constantly rubs the index-finger with its pulp, as if it were attempting to rub off the skin of that member. Frequently there are pain and aching of the extensor muscles of the forearm and wrist from the constant exertion. (See chapter on Feet and Legs, part on Gait.)

The tremors of disseminated sclerosis are also slow, but coarse in character. They are not constant, but are developed upon *intentional movement*, and have a greater amplitude than those of Parkinson's disease (paralysis agitans). Indeed, they may be so coarse as to be choreic in type, or even ataxic. Often threading a needle will be possible for a person with this disease, because it is a short act, while lifting a glass of water will be impossible. The symptoms of disseminated sclerosis are well summarized in the following table drawn up by Charcot.

I. Spinal Symptoms:	
Positive	Tremor on voluntary movements of the extremities—"intention tremor" (arms and head; more rarely of legs). Titubation. Paresis (spasmodic) of the extremities. Contracture, with exaggeration of the reflexes—spastic rigidity.
Negative	No sensory symptoms, or only very slight disturbance. Vesical disturbance none or very slight.
II. Cerebral Symptoms:	
	Dysarthria—slowness of speech; scanning of words. Nystagmus—blank expression. Attacks of vertigo—spasmodic myosis. Transitory amblyopia—white atrophy of the papillæ. Diplopia—associated paralysis of ocular muscles. Mental enfeeblement. Apoplectiform and epileptiform attacks. Difficulty in deglutition.
III. Abnormal or Unusual Symptoms:	
Trophic	Muscular atrophies (amyotrophies), bedsores.
Tabetic	Lightning pains. Romberg symptom. Anæsthetic areas. Vesical and rectal paresis. Gastric crises.

Frequent remission of all the symptoms is characteristic of the malady.

It is not to be expected that all these symptoms will be found in one case. But many of them will occur. Charcot taught that tremor involving the head indicated disseminated sclerosis, and excluded paralysis agitans; but cases of head tremor in the latter disease do occur. (See chapter on Feet and Legs, part on Gait.)

The tremor of mercurial, plumbic, and alcoholic poisoning resembles that of paralysis agitans, save that it is more rapid, reaching nine or ten vibrations per second, and in the case of alcoholic tremor is decreased by a large drink of liquor, while those due to lead and mercury may be relieved in a short time by potassium iodide. Further than this, the tremor of alcoholism is generally worse in the morning.

A point of some importance in plumbic neuritis producing tremor and wrist-drop is the fact that painful sensations are rarely present; in arsenical neuritis, on the other hand, they are often the most prominent symptoms, even preceding the motor disturbance. In mercurial neuritis, on the other hand, tremor precedes all evidence of loss of power, and, finally, may become so coarse as to resemble chorea.

The tremor of general paresis is also rapid, eight or nine per second, and is a very fine tremor, which may be felt only when the arm is extended and the finger rested on the hand of the physician. In other words, the tremor of the hand in general paresis is generally not a predominant symptom, but is elicited when the muscles are put upon a strain. In regard to the fineness of the tremor of general paresis, it should be remembered that it closely resembles that of Basedow's or Graves's disease (exophthalmic goitre (eight or nine per second), since the tremor of this condition is not only equally fine, but generally unseen except when the arm is extended and tips of the fingers rested upon the fingers of the doctor. This tremor has been called the "railroad bridge tremor," because of its fineness and vibratory character. The individual fingers do not separately tremble in Graves's disease.

In post-hemiplegic tremor the trouble is unilateral, there is a history of cerebral disease, and paralysis is present.

Tremor of a very marked character may be due to hysteria, and arises most frequently in those who have been exposed to shocks or accidents. The tremors may occur constantly or only with intention-movements, or be increased in amplitude but not in rhythm on movement. The latter form is known as the "type Rendu," and has a rhythm of seven to nine per second, while the slower hysterical tremor may be four or five per second.

Beyond the state of tremor should be recalled the movements of chorea (see page 77), which may be limited to one arm or hand, and which in their milder forms may be confused with the pronounced movements produced by effort in disseminated sclerosis. The latter are often very arhythmical, and so the choreic movement the more closely resembles them; but those of sclerosis are purposive, while those of chorea are not, since the movement contemplated in chorea is opposed by a contradictory contraction.

General Movements of the Hand and Arm. Aside from the movements of tremor, careful notes should be made of the movements of the hand as a whole, of the co-ordination of its fingers and of the arm governing it. Thus, trembling contractions of the extensor tendons (subsultus tendinum) are a sign of grave and advanced forms of typhoid fever, and picking at the bedclothes (carphologia) is of still graver import (see beginning of this chapter). Inability to write, to play musical instruments requiring the use of the fingers, or to sew, may indicate the rare form of locomotor ataxia involving the upper extremities, so that if the patient is asked to close his eyes and feed himself the fork or spoon misses his mouth through lack of co-ordination, although loss of power may not be present.

Sometimes in locomotor ataxia as the disease becomes advanced paroxysmal twitching of the fingers may come on, or involuntary movements of the fingers occur in association with voluntary movements elsewhere.

In locomotor and Friedreich's ataxia also the movements of the hand are often lacking in co-ordination. The hand may be advanced past the object which the patient desires to grasp, or else falls short of it. On endeavoring to pick up an object the fingers are spread over it like a widespread claw. Generally these ataxic symptoms will be more marked in the other parts of the body and be bilateral, but Ormerod has reported an instance in which only one hand (the left) was involved. This faulty movement of the hand may, however, be due to the fact that the ocular muscles are affected, and the "erroneous projection" due to this cause leads the patient to pass the hand beyond the object reached for. Overdoses of strychnine sometimes cause this symptom of "erroneous projection."

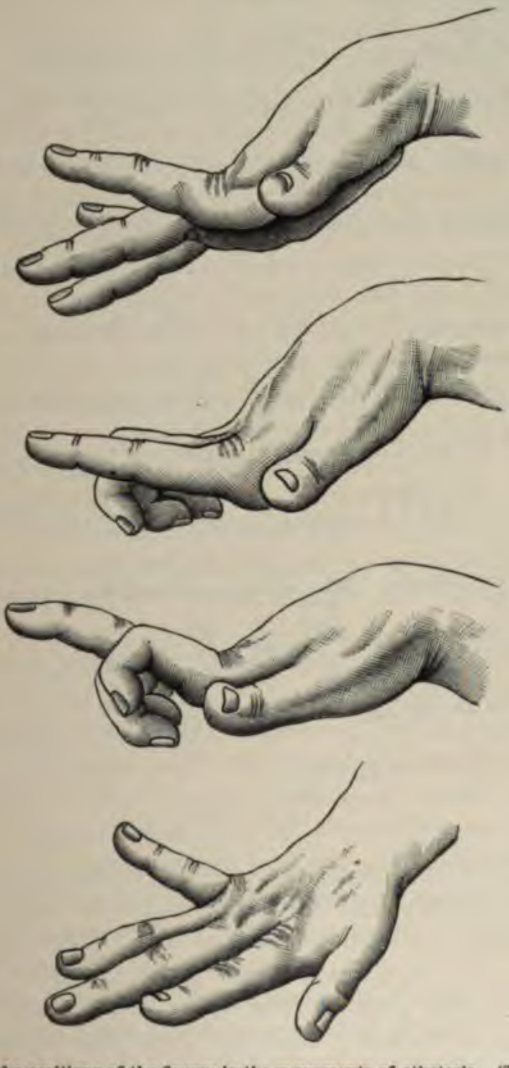
When fibrillary twitchings of the muscles occur and tapping the muscles produces idiopathic muscular contraction, progressive muscular atrophy may be present or profound asthenia.

Sometimes, as the result of infantile cerebral paralysis or from lesions developing in later life, the muscles of the hand are affected by a slow, constant movement, so that the fingers assume curious, constrained, and unusual postures, being moved into extreme or forced extension, flexion or pronation, or supination. (Fig. 30.) This condition is called athetosis, and is separable from chorea in that the movements are slower and limited to the fingers and wrists, the arm escaping.

Very rarely athetoid movements of the fingers occur in advanced spinal tabes (locomotor ataxia), probably as the result of a related lesion, and not from tabes itself.

In this connection mention should be made of "mirror-writing," a curious condition in which the patient writes from right to left instead of left to right. It occurs in some cases of mental feebleness, hereditary or acquired, and rarely in hysteria. "Mirror-

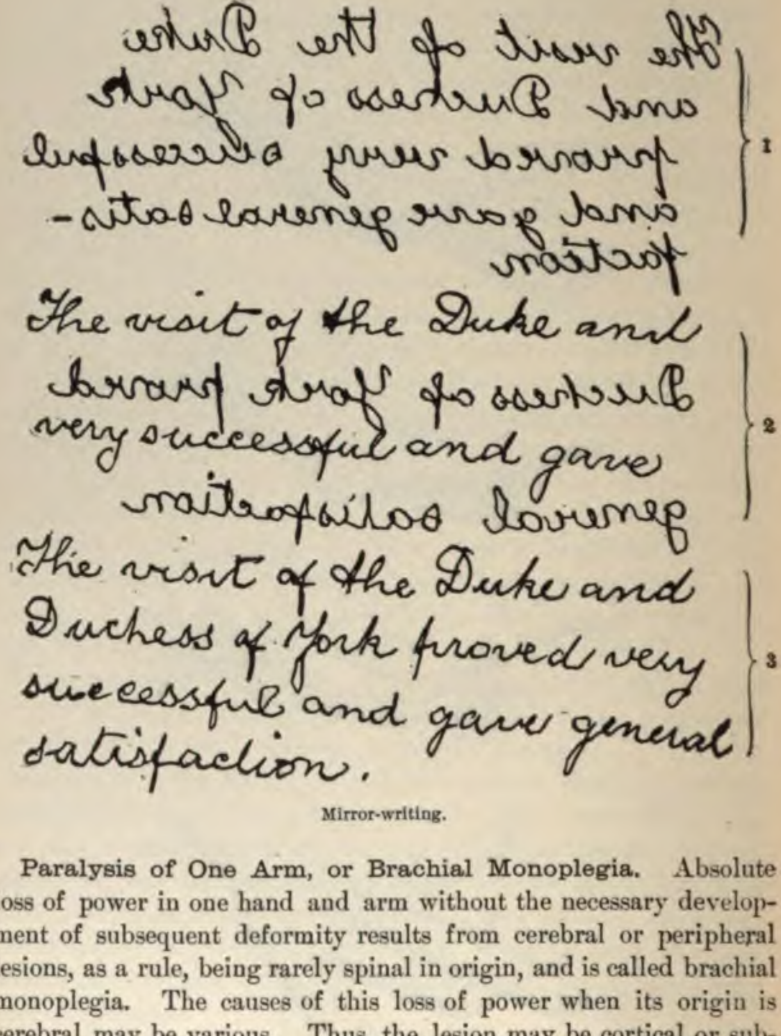
FIG. 30.



Examples of the positions of the fingers in the movements of athetosis. (STRÜMPELL.)

writing" may also be present in cases of cerebral paralysis. The following example of this, taken from a case reported by Clapham, illustrates the character of the handwriting. The patient, a woman of twenty-four years, could write all three ways, but mirror-writing was easiest to her. (Fig. 31.)

FIG. 31.



Mirror-writing.

Paralysis of One Arm, or Brachial Monoplegia. Absolute loss of power in one hand and arm without the necessary development of subsequent deformity results from cerebral or peripheral lesions, as a rule, being rarely spinal in origin, and is called brachial monoplegia. The causes of this loss of power when its origin is cerebral may be various. Thus, the lesion may be cortical or subcortical; that is, in the surface of the brain or in the internal capsule, or between the cortex and the capsule in the corona radiata. As a rule, however, monoplegia is cortical in origin, for below the cortex the motor fibres run so closely together that only a very small lesion can involve one without involving all, and so producing a hemiplegia. These cortical lesions when they do occur are generally, but not always, associated with a convulsive seizure in the paralyzed limb, and Seguin has called this convulsion the "signal-symptom" indicating a cortical lesion. Brachial monoplegia not due to hysteria or neuritis, preceded and accompanied by a convulsion and loss of consciousness, and lacking in signs of involvement of lower nervous centres, is, therefore, cortical, and is generally due to the formation of a clot in the hand and arm centre resulting from injury or from the ordinary vascular causes of apoplexy. In other cases it is due to cerebral embolism or thrombosis, or to the growth of some neoplasm, specific or otherwise, or to a localized meningitis.

The probability of the lesion being an embolism or thrombosis is decreased by the recollection of the fact that the cortex is so well supplied by vessels from the pia mater that paralysis of a centre from lack of blood-supply from such a cause is rare, unless the lesion is subcortical, or, in other words, not deep enough to involve fibres from other centres as they approach each other, and yet sufficiently deep to prevent the tissues from partaking of the nutrient blood-supply from the pia mater as just mentioned. Aside from the discovery of a condition of the internal organs, such as cardiac valvular disease or sepsis, which might cause embolism, the diagnosis between paralysis from hemorrhage and embolism is practically impossible, and this is also true of the paralysis due to thrombosis, except that in cases of thrombosis we often find the presence of general endarteritis or an infectious disease, and the paralysis of thrombosis may be slow and gradual in its onset. If the paralysis rapidly spreads, the lesion is probably due to a hemorrhage.

The history of there having been some sudden cause for an increase in arterial tension, as by muscular effort, and the presence of atheromatous vessels aid us in deciding as to the probability of the lesion being due to a hemorrhage, and the sudden onset, coupled with the symptoms named, makes the diagnosis clear in a certain proportion of cases.

Neoplasms or tumors of the brain producing monoplegia are gradual in their development, accompanied generally by headache, by changes in the optic disks, and sometimes by mental disturbances or pressure-symptoms. A specific history pointing to the formation of a syphilitic tumor is of value in the diagnosis. (See chapter on Headache.)

If brachial monoplegia results from a lesion in the internal capsule, the lesion must be very limited, or, in other words, only large enough to cut off the hand and arm fibres. Tumors and lesions from traumatisms in this area are very rare, and hemorrhages, which frequently cause paralysis by affecting this area, are generally profuse enough to cause hemiplegia—that is, injury of the motor fibres supplying the leg muscles as well. Sometimes, however, a sudden inflammatory process is set up in the tissues surrounding a tumor, and this may precipitate sudden paralysis.

Although the onset of a monoplegia due to cortical, subcortical, or capsular causes is sudden, the reactions of degeneration do not come on for a long period of time in such cases, because the muscles in the paralyzed area are still connected with the trophic centres in the cord, and this affords us a valuable point in differential diagnosis.

Sometimes a suddenly developed monoplegia affecting the arm comes on as a manifestation of hysteria, and follows the type of true cerebral hemorrhage so closely as to almost defy diagnosis. This condition may be accompanied by hysterical œdema, the hand becoming puffy and swollen. The presence of a neurotic temperament and other hysterical signs, coupled with the prompt development of contractures, and the fact that the muscles do not rapidly waste, point to the cause of the loss of power in some cases, and this is emphasized if the presence of hysterical anæsthesia of the skin can be discovered. Further, if the hand is affected, Patrick asserts that in making an attempt to grasp an object the thumb and forefinger are chiefly used; but if the object is placed suddenly in the ulnar part of the hand, the remaining fingers can grasp it easily. (See chapter on the Skin for additional hysterical symptoms.)

Brachial monoplegia is very often the result of injury to the brachial plexus or to some of its important branches. The symptoms consist in heaviness or numbness of the arm with more or less loss of power. The motions of the arm which are particularly affected are usually abduction and elevation, which movements depend upon the circumflex nerve. If the power of extending the arm is lost, the loss depends upon paralysis of the musculo-spiral, which supplies the triceps; whereas if the power to flex the forearm is lost, there is paralysis of the musculo-cutaneous, which is the supply of the brachialis anticus and biceps. If the supinator longus is involved, the musculo-spiral is also affected.

When brachial monoplegia depends for its existence upon primary brachial neuritis, there is pain in the wrist and hand at first, or on the scapula and in the axilla, thence radiating down the arm. This pain is constant and dull, and now and then excruciating, and is made worse by movement, even when the loss of power is comparatively slight. Sometimes, on the other hand, when the neuritis is septic in origin, it may start in the ulnar nerve and gradually extend up to the plexus. In still other cases brachial monoplegia may depend upon fracture or dislocation of the head of the humerus, and in such a case the paralytic symptoms are apt to be very well developed. The musculo-spiral nerve is often paralyzed by fracture of the humerus, and this results in paralysis of the muscles of the back of the arm and forearm and back of the hand, and loss of sensation in the skin covering these parts.

In all cases of brachial monoplegia due to peripheral lesions, as in severe neuritis, we find that atrophy of the muscles comes on very rapidly owing to the cutting off of the muscles from their trophic centres in the spinal cord.

Sometimes in locomotor ataxia the peripheral nerves seem quite as much involved as the spinal cord, and symptoms precisely like the paralysis of a toxic neuritis develop. Thus, Strümpell has reported cases of musculo-spiral paralysis from this cause, and Remak and Hirt record cases in which the median nerve has been affected, so that not only loss of power but wasting of the muscles has resulted. This is particularly the case if the muscles are much used in the daily pursuits. The ulnar nerve may also be affected. Such cases are separated from pseudo-tabes, due to neuritis, by the pupillary reflexes and other pathognomonic ataxic symptoms. (See chapter on the Feet and Legs, and that on the Eye.) Widespread muscular atrophy of the arm sometimes takes place in locomotor ataxia as a result of a coincident neuritis.

There are still to be considered two comparatively rare forms of brachial monoplegia of the plexus type, namely, that due to pressure of growths in the neck or axilla, and brachial paralysis of the upper arm type, sometimes called Erb's paralysis. This latter form occurs from paralysis of the fifth and sixth cervical nerves or their roots. In adults this commonly results from blows or heavy weights striking on the shoulder, and in infants from pulling on the neck in difficult labor. As already said, it is an upper arm palsy, and is due to the loss of nerve-supply to the deltoid, biceps, brachialis anticus, and supinator longus and brevis, and the supra- and infra-spinatus muscles. The adult form is often associated with anæsthesia and is persistent. In infants it is often temporary, and sensory symptoms are commonly absent.

When the lower arm is paralyzed as the result of trouble in the brachial plexus, the lesion is in the nerves arising from the seventh and eighth cervical and first dorsal roots, and the muscles affected are the triceps, the flexors of the wrist and fingers, the pronators of the hand, the extensors of the fingers, and the muscles of the hand. The arm can still be raised by the deltoid and the forearm flexed on the arm by the biceps.

When there is wasting with paralysis of the thenar, hypothenar, and interossei muscles, not due to progressive muscular atrophy, with anæsthesia in the arm and forearm in the part supplied by the ulnar nerve, and in addition myosis on the side of the lesion, with sluggish pupil, retraction of the eyeball, and partial closure of the lids, there is probably a lesion of the first dorsal root of the brachial plexus and the communicating branch of the second dorsal. The cause may be neuritis or pressure by a tumor. This form is sometimes called "Klumpke's paralysis."

(For a description of the areas involved in the spinal cord, which cause loss of power in the arms and legs, see chapter on Feet and Legs, part on Paraplegia, and tables of localization in that chapter, also plates in chapter on Skin.)

The presence of bilateral brachial monoplegia should always make the physician suspicious of lead-poisoning or crutch-paralysis.

Apparent brachial monoplegia, in reality a syphilitic pseudo-palsy, has been described particularly by Parrot. A child apparently perfectly well, and but a few weeks old, suddenly loses the power of its arm, so that the member hangs like a flail. No wasting takes place, no degenerative reactions occur, but there may be some pain and crepitation on moving the arm. The cause of these symptoms lies in the fact that there has been a separation of the epiphyses from the shafts of the bones, with consequent helplessness. Sometimes general paralysis of the extremities arises from the extension of the disease to other limbs. The prognosis as to life is bad.

It yet remains for us to discuss the paralysis of several important groups of the muscles of the arm. If the forearm cannot be flexed, there is loss of power in the biceps and brachialis anticus, and to some extent in the supinator longus; and as the first two muscles are supplied by the musculo-cutaneous, and the third by the musculo-spiral, such a failure in flexion shows paralysis of these fibres.

Paralysis of the extensors of the forearm, wrist, and hand, and of extension of the elbow, with wrist-drop in consequence, and flexion of the tips of the fingers, is due to disease affecting the musculo-spiral nerve, but the fingers can still be partly extended through the action of the interossei and lumbricales, provided the tips are flexed. The back of the hand and wrist become unduly prominent after a short time because of the forced flexion of the hand and rapid wasting of the extensors. In most cases the supinator longus, which supinates the forearm after it is pronated, is paralyzed. When the ability to pronate the forearm is greatly impaired, and the thumb is extended and abducted, so that it cannot be brought in contact with the tips of the fingers, the trouble is probably paralysis of the median nerve, and this is confirmed if all the phalanges are paralyzed except the first.

If the arm cannot be moved outward, away from the body, there is paralysis of the deltoid supplied by the circumflex nerve. In this connection attention should be called to the loss of power with wasting of the muscles seen after direct blows on the muscle or after injuries to the joint, sometimes called "joint-palsies."

Brachial Paræsthesia. Disturbances of sensation in the hand and arm consist in anæsthesia, analgesia and numbness, tingling, and pain. The area of these sensations depends upon the nerve-trunks involved, and to some extent upon the degree of involvement. Thus, if the function of the nerve is merely impaired, the sensation may be that of tingling or pain; if still further impaired, the sensation may be that of numbness; and if the sensory fibres be totally destroyed or paralyzed, absolute anæsthesia and analgesia may be present. (See Anæsthesia, chapter on Skin.)

CHAPTER III.

THE FEET AND LEGS.

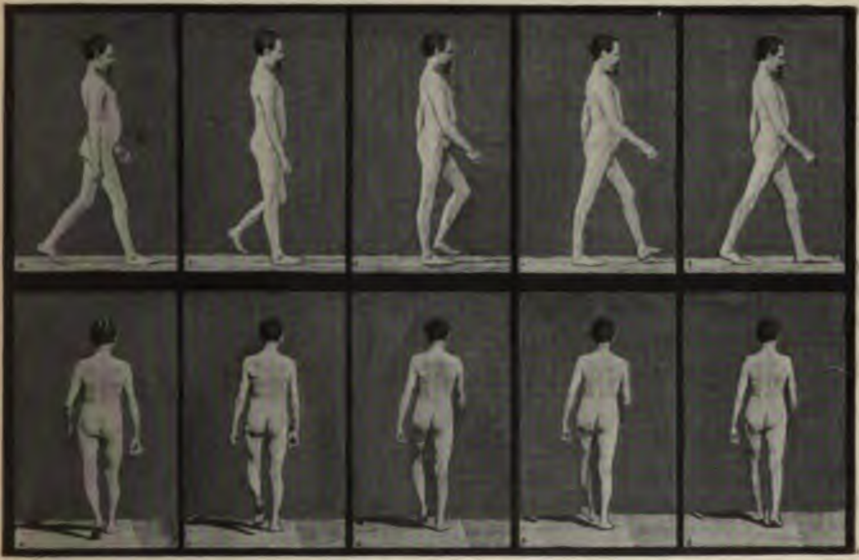
The general appearance of the feet and legs when clothed—The gait—Spastic paraplegia—Paraplegia without spastic contraction—Crural monoplegia—Deformities of the feet and legs—The joints—Alterations in the nutrition of the feet and leg aside from a change in the muscles.

As the physician sees a patient approaching him, he can often gain information as to the ailment from which the man is suffering by noticing his gait and the appearance of the legs and feet, for, while the gait varies greatly in normal individuals, in some diseases it is so typical that he who runs may read the diagnosis. A glance at the feet revealing one foot more loosely covered than the other, or a slit in the shoe, or a very loose lacing, will point to the presence of some inflammatory or dropsical swelling, which forces the patient to give it room; and if the legs of a man of ordinary build look swollen and fill the trowsers tightly, while a glance at his face reveals that it is puffy, rather than one which is obese, dropsy still more widespread is probably the cause.

Gait. Aside from local injuries causing a lame gait, which will be found discussed in a book on surgical diagnosis by the writer's friend, Dr. Martin, we find that gout, rheumatism, and sciatica are the common causes of a limping gait, arising from trouble in one leg, and that in such cases there is a pained expression of the face at each movement, which shows the suffering that walking causes. The gait of such a patient is slow and cautious, and he is apt to rest every few steps, bearing his weight at such times chiefly on the well leg, or, by means of his hands, upon chairs or tables that may be near. Aside from the alterations of gait produced by these causes, we see very typical gaits produced by locomotor ataxia, pseudo-locomotor ataxia (peripheral neuritis) due to alcoholic or lead-poisoning, syphilis, or peripheral neuritis arising from other causes, Friedreich's ataxia, general paresis, chronic myelitis, lateral sclerosis, acute poliomyelitis, pseudo-muscular hypertrophy, cerebral infantile palsy, multiple sclerosis, paralysis agitans, cerebellar disease, organic and hysterical hemiplegia, and osteomalacia, and the gaits caused by rickets and other bony defects.

In locomotor ataxia the gait is unsteady and waveringly uncertain, resembling that of a blindfolded person who is told that he is approaching some inequality in the floor. The patient continually seems to be feeling for the ground with his feet, and carefully picks his way along a perfectly smooth surface in a labored fashion, using a cane to help him both in the way of support and of feeling the ground. If he looks up from the ground while walking, he sways suddenly and may fall; and if prevented from returning his eyes to the pavement, almost surely falls if no aid is given him. (Fig. 32.)

FIG. 32.



Gait in a case of locomotor ataxia. From instantaneous serial photographs of a patient of Dr. Dercum, made simultaneously from two different points of view by Muybridge.

The gait of pseudo-tabes is sometimes identical with that just described, is usually associated with a history of alcoholic excess, and is due to multiple neuritis. In a majority of the cases, however, it is distinctive, and has been called the "steppage" gait. The foot is thrown forward and the toe is raised so that the heel first strikes the ground in much the manner adopted when one attempts to step over some obstacle. Sometimes this gait is found in cases of arsenical neuritis and that due to lead, but in alcoholic tabes there are generally mental symptoms associated with this gait, while in lead-poisoning the pathognomonic signs of this condition, such as the blue line on the gums and wrist-drop, when combined with the history, clear up the diagnosis. It must not be forgotten, however, that the differential diagnosis of tabes from pseudo-tabes is sometimes very difficult, and as Dana has well said: "When Déjèrine described as locomotor ataxia a case which now appears to have been one of alcoholic peripheral neurotabes, when Buzzard has diagnosticated as true spinal tabes a case of post-diphtheritic ataxia, when Seligmueller mistakes a case of wall-paper-poisoning for one of true spinal tabes, we may easily suppose that errors have been made by many others."

The important symptoms which point to true locomotor ataxia are the swaying of the body when the eyes are closed (Romberg's symptom), the loss of knee-jerk (Westphal's sign), the history of gastric, laryngeal, or vesical crises, the presence of numbness in the feet, the slow onset of the disease, and the absence of any history of exposure to the causes of neuritis just named. Additional diagnostic points are the inability of the patient to stop and turn quickly and steadily at the command of the physician, and Fränkel states that in many cases of this disease the sensation of passive motion at the joints is impaired. To determine the presence of this symptom the toe is grasped by the thumb and forefinger, and moved very slowly and gently so as not to disturb the rest of the limb. The patient must be blindfolded, and in ataxia fails to appreciate that passive joint movement has been made. If all these signs are present, and are combined with that important symptom, the Argyll-Robertson pupil, the diagnosis is practically certain.

Grube has, however, recently reported three cases of diabetes mellitus producing a pseudo-tabes due to neuritis which had the Argyll-Robertson pupil, and in addition attacks of abdominal pain like the crises of true ataxia.

Another sign of locomotor ataxia is undue relaxation of the muscles, which has been called hypotonus. This is a point much insisted upon by Fränkel. He points out that if a healthy man be placed in a horizontal position on a couch, he cannot raise the leg very high if the knees be kept extended. On the other hand, if he has locomotor ataxia, even with the knee fully extended, he can raise the leg to a greater angle with the plane of the body, amounting to 60, 80, or 100 degrees; whereas in health he cannot raise it to an angle greater than 30 to 50 degrees. This is due to the fact that the semimembranosus and semitendinosus resist the movement in health;

whereas in locomotor ataxia they are so relaxed that this resistance does not occur. Again, because of hypotonicity of the quadriceps, it may be possible in such patients to flex the knees to such an extent that the heel can readily touch the buttocks in voluntary motion. So, too, the thighs when the legs are flexed may be so widely abducted that the knees come in contact with the couch, because of relaxation of the adductors.

The following table from Peterson's article in Dercum's *Nervous Diseases* shows very clearly and comparatively the symptoms of the first, second, and third stages of true locomotor ataxia.

THE STAGES OF TABES DORSALIS.

Initial Period.	Second Stage.	Final Station.
Inco-ordination, but no change of gait.	Greater inco-ordination, and marked ataxic gait.	Cannot walk because of ataxia.
Numbness of the feet.	More marked anæsthesias.	Extensive anæsthesia.
Shooting-pains in the legs.	Pains worse.	Pains less.
Diminished or lost knee-jerks, one or both.	Lost knee-jerks.	Lost knee-jerks.
Sluggish or lost pupillary reflex to light.	Lost pupillary reflex to light and myosis.	Lost reflex to light, myosis, paralysis of accommodation.
Weakness of sexual function.	Impotence.	Impotence.
Transient diplopia ; transient ptosis	Ocular palsies rare, or marked ophthalmoplegia.	Ophthalmoplegia.
Sluggish micturition.	Increased vesical weakness.	Catheterization needed.
Optic atrophy.	Optic atrophy rarely develops.	Blindness.
Trophic changes in the joints.	Trophic changes not so common.	More marked if they began in early stage.
Hemiatrophy of tongue.	Deafness.	Increased.
	Laryngeal and visceral crises.	Not so common.
	Girdle sensation.	Unnoticed.

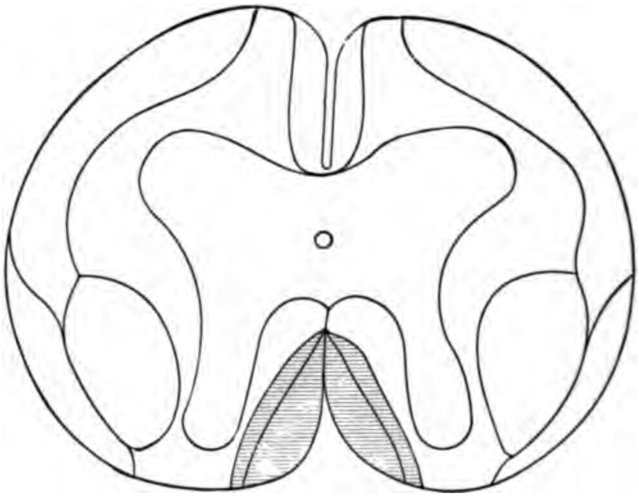
In neuritis causing pseudo-tabes we have a history of rapid onset of the symptoms, paralysis and wasting of the muscles, and *an absence* of vesical symptoms and of the Argyll-Robertson pupils.

From a prognostic and therapeutic stand-point it is interesting to note that about 80 per cent. of all cases of tabes dorsalis are syphilitic. (Duckworth.)

Reflex action is decreased and the gait altered in locomotor ataxia, because, though the motor tracts are open, the sensory tracts in the nerves, the posterior nerve-roots, and the posterior columns of the cord are diseased. (Fig. 33.) For these reasons the reflex arc is destroyed and the co-ordination of the muscles lost. The patient cannot tell how to use his muscles unless he can see them and co-ordinate them by the aid of the eye. The sensations of formication or numbness are also due to these sensory lesions. (For descriptions of motor and sensory tracts of the spinal cord, see early part of chapter on Hemiplegia, and the chapter on the Skin.)

Sometimes not only the gait, but the entire set of the ordinary symptoms of locomotor ataxia are aped by hysteria so closely that a diagnosis may be almost impossible, but the Argyll-Robertson pupil, the lost knee-jerks, and the optic atrophy will not be present if hysteria be the cause of the symptoms. On the other hand, Romberg's symptom may be marked to an extraordinary degree. The patient who is hysterical, in falling nearly always falls the same way, keeping her frame stiff like a board. (See chapter on Eye for differential ocular symptoms.)

FIG. 33.

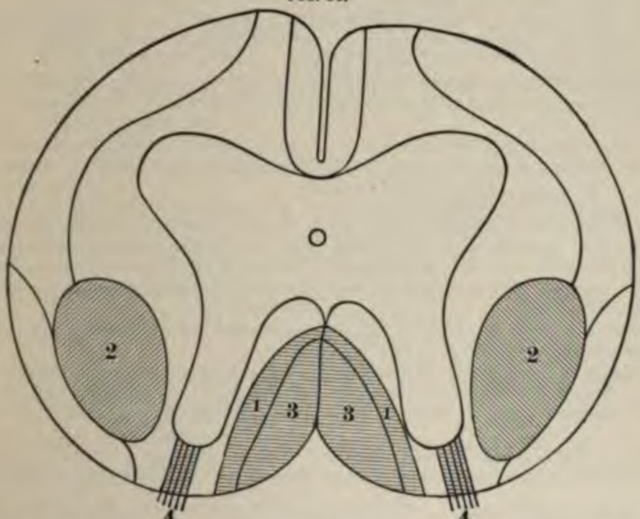


Showing the areas of the cord involved in locomotor ataxia. The shading includes both the column of Goll, the inner, and that of Burdach, the outer. It is to be remembered that the lesions of locomotor ataxia are found in the peripheral nerves as well.

The feebleness of the limbs, the reflex iridoplegia (Argyll-Robertson pupil), and the ataxic gait sometimes seen as the chief manifestations of *general paresis* may cause an error in diagnosis in favor of locomotor ataxia, but careful examination will reveal mental feebleness in the paretic case, or at least evidences of delusions, and if the disease is at all advanced there will be a history of the patient having had convulsions or apoplectiform attacks. Sometimes there will be found present in paretic dementia increased knee-jerks and many of the symptoms of ataxic paraplegia, but the associated mental failure and fine intention-tremor of the hands decide the diagnosis in favor of paretic dementia.

In Friedreich's ataxia the gait is peculiar. The legs are widely separated and moved in an uncertain, hesitating manner, and if the feet are placed close together and the patient is told to stand still, swaying at once develops. If the eyes be closed, the swaying may greatly increase. The movements of the arms are inco-ordinated. These symptoms, which to a certain extent simulate true locomotor ataxia, are associated, as a rule, with others which separate the two affections, for in this disease the symptoms often come on in very early life, there is sometimes nystagmus, usually a history of heredity, there are a slow and jerky articulation, scoliosis, and talipes equino-varus, but there is no Argyll-Robertson pupil. (Fig. 34.)

FIG. 34.



Showing the spinal areas chiefly involved in Friedreich's ataxia. The areas are the column of Burdach (1); the lateral pyramidal tracts (2); the columns of Goll (3); the posterior nerve-roots (4).

The following table shows the differential points between locomotor ataxia and Friedreich's ataxia:

LOCOMOTOR ATAXIA.	FRIEDREICH'S ATAXIA.
Argyll-Robertson pupils.	No Argyll-Robertson pupils.
No nystagmus.	Present late in disease.
Painful crises.	Crises usually absent.
Intellect unimpaired.	Becomes impaired.
Gait ataxic.	Cerebellar ataxic gait.
Speech normal.	Speech halting.
No head tremor.	Head tremor present.

Friedreich's ataxia must be separated from another rare disease in which the gait is ataxic and the disease hereditary, namely, hereditary cerebellar ataxia, in which we have the following symptoms not seen in Friedreich's disease, namely, normal or exaggerated knee-jerks, Argyll-Robertson pupils, and a beginning of the malady after twenty years of age. The following table, compiled by Collins, of New York, gives the differential points between these diseases :

HEREDITARY SPINAL ATAXIA. *Friedreich's disease.*	HEREDITARY CEREBELLAR ATAXIA.
1. Gradual impairment of co-ordination, first in legs, afterward in arms. Later in the disease the patient may reel, as if under the influence of alcohol. A quick backward and forward balancing movement.	1. Gait: uncertain, reeling; gait of one inebriated. Patient frequently walks with body bent forward and head thrown backward, and the feet wide apart. Does not have to watch the feet.
2. Station: closure of eyes, as a rule, increases the unsteadiness; this may be absent.	2. Station: Romberg symptoms absent.
3. Titubation of upper extremities very uncommon. Irregularity in voluntary movements of arms and fingers.	3. Titubation and inco-ordination and loss of dexterity in the upper extremities. Choreiform movements exaggerated on voluntary effort; "intentional."
4. Frequently jerky, irregular movements of head and neck. Sometimes like an irregular tremor.	4. Not infrequently oscillations or jerky movements of the head, less often of the trunk.
5. Mimetic muscles do not show ordinarily overcontraction.	5. Exaggerated contraction of the mimetic muscles on speaking.
6. Ataxia is not so great when the patient is lying down.	6. Ataxia is very much less, or disappears when the patient is lying down, but the inco-ordination persists.
7. Affection of speech may be absent; when it does occur is a late symptom, and consists of an eliding of syllables and an occasional hesitation.	7. Speech: hesitating, abrupt, explosive, ataxic, defective.
8. Nystagmus is a very common late symptom, but it may be lacking.	8. Eyes: twitching of the eyeballs very common, but not nystagmus. Optic atrophy, progressive choroiditis, paralysis or paresis of the external recti sometimes.
9. Myotatic irritability is lost. Knee-jerks may be present in the beginning of the disease, but they soon disappear. Ankle-clonus is never present.	9. Myotatic irritability increased; reflexes exaggerated, such as knee-jerks; often ankle-clonus.
10. Mentally normal. Very rarely any defect.	10. Mental shortcomings varying from slight psychical disturbances up to a considerable degree of dementia.
11. Deformities of the extremities, such as *pied bot* and spinal curvature, very common.	11. Deformities of the extremity and spine, such as *pied bot* or scoliosis, do not occur or are very rare.

Hereditary cerebellar ataxia may also be confused with disseminated sclerosis, as is shown in the following table prepared by Stieglitz. Hereditary cerebellar ataxia is, however, much more rare than sclerosis. The differential points are shown in italics.

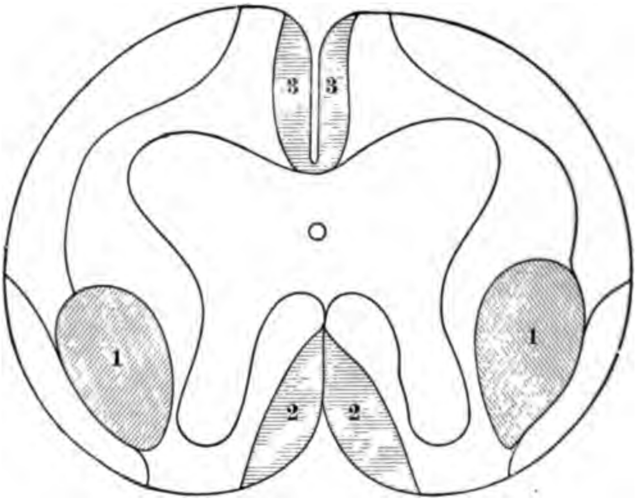
HEREDITARY CEREBELLAR ATAXIA.	DISSEMINATED SCLEROSIS.
1. Gait: ataxic, groggy; feet wide apart.	1. (*a*) Spastic paraplegia; feet close together. (*b*) Ataxic, groggy; feet wide apart. (*c*) Ataxic paraplegia (*a* + *b*).
2. *Station: Romberg's symptom absent.*	2. *Romberg symptom may be present.*
3. Arms: ataxy and some intention tremor.	3. Intention tremor; sometimes ataxy.
4. Oscillations and jerky movements of the head and trunk.	4. Oscillations and jerky movements of the head and trunk.
5. Exaggerated contractions of facial muscles during speaking.	5. Twitching in facial muscles during speaking.
6. *Speech: hesitating and abrupt, or simply monotonous.*	6. *Laborious, scanning,* or monotonous speech.
7. Eyes: jerky nystagmus; optic atrophy, contracted field of vision. The external recti muscles may be paretic or paralyzed.	7. Jerky nystagmus; optic atrophy, contracted field of vision; ocular nerve palsies.
8. Myotatic irritability increased, knee-jerks exaggerated, ankle-clonus; contractures and muscular rigidity.	8. Myotatic irritability increased; knee-jerks exaggerated, ankle-clonus; contractures and muscular rigidity.
9. Mental impairment in varying degrees.	9. Mental impairment in varying degrees.
10. *Vertigo sometimes.*	10. *Vertigo common.*
11. *Vesical functions rarely affected.*	11. *Vesical functions more frequently disturbed.*
12. *Apoplectiform seizures do not occur.*	12. *Apoplectiform seizures occur in a small proportion of cases.*
13. *Heredity common.*	13. *Heredity uncommon.*

Stieglitz has also pointed out that in certain cases of acute disseminated myelitis and encephalomyelitis following the acute infectious diseases, the symptoms of an acute or subacute multiple sclerosis are presented, more especially the intention-tremor, the increased reflexes, and the scanning speech. The disease may ultimately form the basis of a typical chronic insular sclerosis with its recurrent attacks, etc. It may, however—and this is a point of importance—subside after a shorter or longer period and end in recovery.

In chronic myelitis in the early stages, while motion is still preserved the gait is typically that of feebleness, and the legs respond slowly to the cerebral desires, being dragged along after the patient, who leans forward, supporting some of his weight on crutches or canes.

If the lesions of the disease involve the lateral pyramidal tracts to a considerable extent, the gait is somewhat spastic, while if the sensory fibres are much involved it may be like that of ataxia. Under these circumstances the attitude and gait of a patient are sometimes a combination of those of lateral spinal sclerosis (spastic paraplegia) and locomotor ataxia. In some instances the spastic symptoms are more marked, in others the signs of locomotor ataxia are more prominent. This condition is called ataxic paraplegia, and in it we find the exaggerated knee-jerks of lateral sclerosis associated with the swaying of the body (Romberg's symptom) of ataxia. Ankle-clonus is also present. The crises of locomotor ataxia do not occur, and the Argyll-Robertson pupil is usually not present. (Fig. 35.)

FIG. 35.



Showing areas of spinal cord involved in ataxic paraplegia, which is practically a combination of locomotor ataxia and lateral sclerosis. 1. Lateral or crossed pyramidal tracts. 2. Posterior columns of Goll and Burdach. 3. Direct pyramidal tracts or Türck's columns.

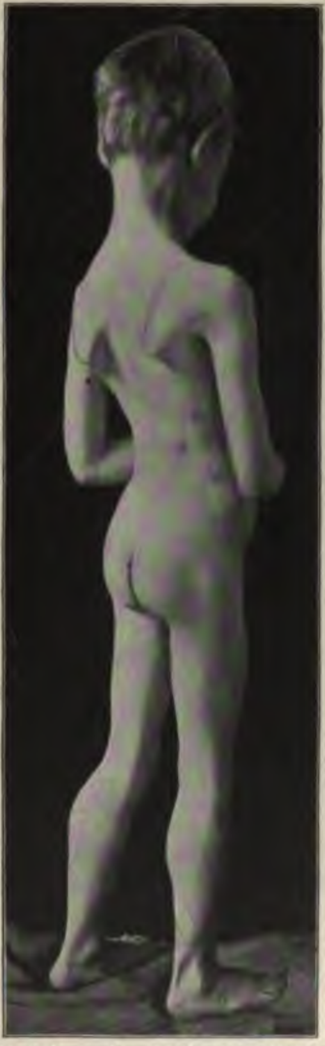
In lateral sclerosis the gait is typically spastic, the legs being rigid from the hip-joint down, and the toe being dragged in a semicircle from behind forward.

When the gait of a young child is stumbling, or the leg or legs are dragged after it, or the ankles bend so that locomotion is impossible, the probable diagnosis is that the cause is acute poliomyelitis. (See Paralysis of Leg.)

In pseudo-muscular hypertrophy there is a peculiar waddling gait, a tendency to stumble, the body is usually bent forward, and there is difficulty in getting up from the floor and on going up and down stairs. The patient in all his movements shows a marked loss of power in the legs with a great apparent increase in the size of the muscles in the legs. (Fig. 36.)

The gait of pseudo-muscular hypertrophy is sometimes closely reproduced in children suffering from severe rickets, and the other features of the case which may mislead the physician are that the child, if fat, will have bulging legs, as if the muscles were hypertrophied, and lordosis due to spinal weakness. In the rickety case, however, the knee-jerk is preserved, and in the case of pseudo-muscular hypertrophy it is lost.

FIG. 36.



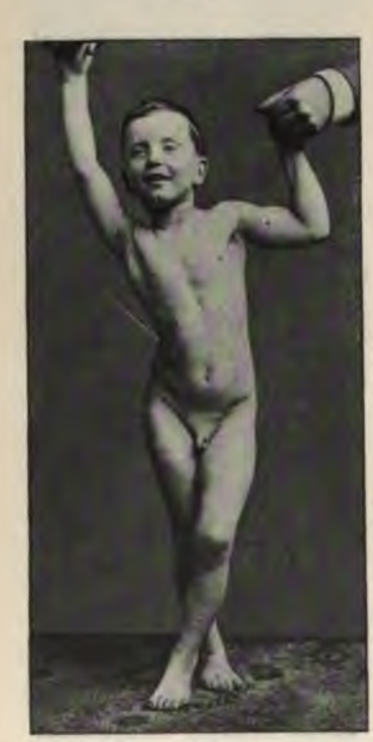
Typical pseudo-muscular hypertrophy. (DERCUM.)

The gait of a child suffering from infantile cerebral paralysis is quite characteristic. In the first place, it is spastic, and the patient walks on the toes, or in some cases club-foot develops. The heels are everted and the toes turned inward, the knees being so closely approximated that the clothes may become worn between them from the rubbing. So great is the extension of the legs that the toes are very apt to drag, and, finally, the adduction spasm may be so great that the legs overlap each other as walking is attempted. (Fig. 37.)

The gait in disseminated sclerosis is often markedly spastic and paretic —that is, stiff and feeble, and may in the early stages of the disease closely resemble that of spastic paraplegia due to lateral sclerosis. When the patient attempts to pick up a small object with his fingers there are tremor and oscillation of the hand. Scanning speech and nystagmus develop later on in these cases. It is, however, important to remember that multiple cerebro-spinal syphilis may closely simulate multiple, or disseminated, sclerosis.

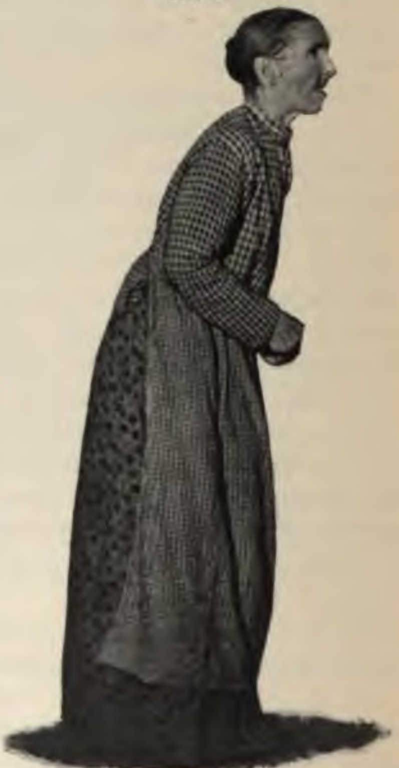
Sometimes they may be differentiated by the fact that in disseminated sclerosis there is apt to be paræsthesia, whereas in syphilis there is more apt to be pain. An important differential symptom is that nystagmus is rare in syphilis, common in disseminated sclerosis, and ocular palsies are common in syphilis, rarely so severe in sclerosis, so that complete oculo-motor palsy with ptosis and squint would be more likely syphilitic than sclerotic. (See chapter on the Eye.)

FIG. 37.



Spastic paraplegia; crossed-legged progression. (From a patient of Dercum's in the Jefferson Medical College Hospital.)

FIG. 38.



Side view of a case of paralysis agitans, showing forward inclination of trunk. Tendency to propulsion. (DERCUM.)

In paralysis agitans the patient's gait is hurried because, from the bent-over position of his body, his centre of gravity is too far forward, and he runs to keep up with it. This is called festination. The gait is also somewhat trotting or toddling. (Fig. 38.) (See chapter on Hands and Arms, part of on Tremor.)

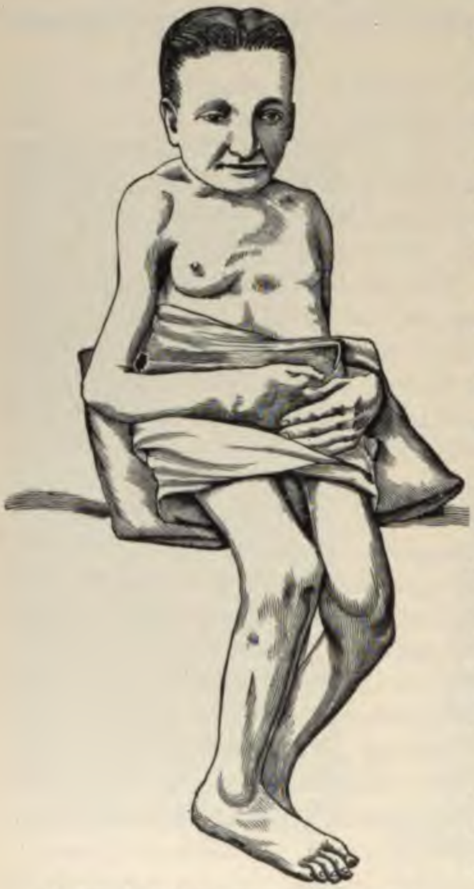
In cerebellar disease the gait may closely resemble that of a drunken man, and the patient has the greatest difficulty in keeping from sheering off to one side as he walks, swaying, too, from side to side (cerebellar titubation). The middle lobe of the cerebellum is usually affected; but Nothnagel asserts that, if these symptoms are associated with paralysis of the oculo-motor nerves and other symptoms of brain-tumor, there is a growth in the corpora quadrigemina.

In hemiplegia the gait is peculiar in the dragging along of the paralyzed limb by a peculiar outward swing, which soon wears away the sole of the shoe on the inner side near the ball of the foot. It is sometimes called a mowing gait, because the leg sweeps around in a half-circle. Very often the shoulder opposite the paralyzed side is raised in order to tilt the pelvis on the paralyzed side, so as to make circumduction easy. This gait is to be clearly separated from that due to hysterical paralysis, for in this condition the leg is dragged after the body without this outward swing. It is dragged along like the broken hind limb of one of the lower animals, or is shoved forward and the well foot drawn after, the reverse of what happens in organic paralysis. The footsteps of the hysterical paraplegic are, moreover, apt to be careful and mincing. Further, the loss of power is usually left-sided, and associated with characteristic hysterical anæsthesia (see chapter on Skin), and often with areas of hyperæsthesia. Again, in the gait of hysterical paralysis the patient is apt to be excessively laborious in her progress, and will exhaust her muscles in her strained movements. An altered gait due to irregularly distributed paralysis of groups of muscles is nearly always hysterical, and sometimes the patient who has hysterical loss of power will suddenly fall through giving way of her knees.

A condition of the gait and station of the patient varying from normal, which occurs most commonly in hysteria, consists in an inability to co-ordinate the movements of the muscles of locomotion or those used in standing. This is called "astasia abasia." It is in reality a form of ataxia often developing only when the patient attempts to walk. There is no loss of power in the legs, but an inability to use them regularly or with power while walking, although if the patient be made to lie down the movements of the limbs as made in walking can be performed perfectly. The knee-jerks are rarely lost, and in addition the general symptoms of hysteria can nearly always be found. The body often reels to and fro, and occasionally the muscles seem to be somewhat spastic. This symptom generally follows some severe shock, and is most commonly seen in young persons, usually young women.

In osteomalacia there is increasing difficulty of walking, partly due to pain and partly to muscular weakness. The gait is hobbling, tottering, and is made up of short and evidently painful steps, "the pelvis and leg being jerked forward as if in one piece." The kyphotic deformity of the spine, muscular tenderness, and lateral compression of the chest and pelvis, with distortions of the limbs, aid in making the diagnosis. (Fig. 39.)

FIG. 39.



Allison's case of osteomalacia. (*Edinburgh Medical Journal.*)

The gait of rickets is only peculiar when curvature of the limbs or spine destroys the normal posture of the body or interferes with the movements of the limbs, but it is nearly always more or less waddling.

We come then to a study of the nervous causes producing these changes in gait. Aside from conditions of the bones and joints we may have loss of power affecting the muscles of the lower extremities: either on both sides, in which case we have a condition called paraplegia; of one lower limb, a condition called crural monoplegia; and of groups of muscles, resulting in localized palsies. These paralyses often produce deformities, as will be shown shortly.

Paraplegia. Given a case of paraplegia, or paralysis of the lower extremities, what may be its cause? It may arise from a cerebral lesion, which is very rare, except in children, when it is common,[1] and if cerebral it must depend upon a lesion on both sides of the cerebral cortex or in each capsule; that is to say, there must be present a lesion in the leg-centres on both sides of the cortex or in the fibres going to the legs through the internal capsules. Much more commonly the lesions causing paraplegia are in the spinal cord, very rarely this symptom is due to involvement of the nerve-trunks on both sides, after they have left the cord, and sometimes it is caused by hysteria and reflex irritation.

When paraplegia occurs in a young child it is due in a great majority of the cases to caries of the vertebræ, and the pressure so produced does not necessarily depend upon compression by the bones, but by the inflammatory exudate.

The *spinal* lesions giving rise to paraplegia of the lower extremities are numerous, and are perhaps best grouped in the following table of Bramwell:

1. Organic disease . . .	Inflammation of cord	Medullary.
	Softening " "	
	Hemorrhage " "	
	Tumors " "	
	Meningitis " "	Meningeal.
	Meningeal hemorrhage	
	Injuries	
	Tumors	
	Caries of bone	Osseous.
	Tumors of bone	
2 Functional . . .	Hysterical.	
	Reflex.	
	Malarial and anæmic.	
	Dependent on idea.	

SPASTIC PARAPLEGIA. The paraplegia of cerebral infantile paralysis is spastic, and follows difficult labors or injuries to the child before or after birth. Contractures nearly always ensue, and exist chiefly in the adductors of the thighs, so that the attitude is very characteristic. (Fig. 40.) Epileptic convulsions very often complicate these cases. Often these paraplegias are not manifested for some months, or even longer, after birth. In many cases they are first noticed when the child attempts to walk.

Cerebral spastic paraplegia in infants also sometimes comes on in cases of so-called arrested development. Such infants present no abnormality for the first few months of life, then cease to develop in mental brightness, fail to recognize the nurse or mother, cease to play, gradually lose their vision, and develop nystagmus. Death usually takes place in one or two years at the latest. Convulsions do not occur in this state, but tremors are often present in the arms. There is no history in such cases of difficult labor or premature delivery.

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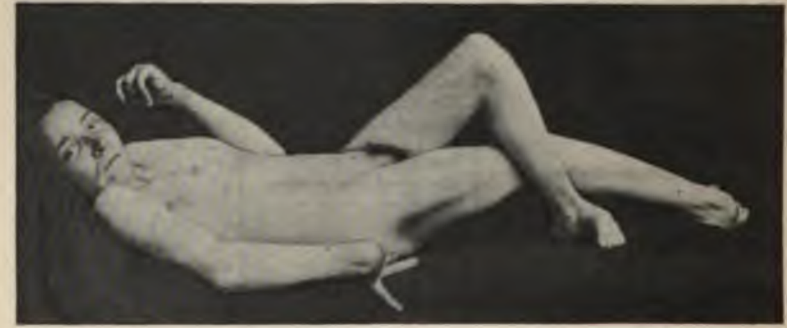
[1] Such an occurrence in adults is very rare, but it is quite common in young children, as many as 14 per cent. of the cases of infantile cerebral palsy being paraplegias. (SACHS.)

Closely allied to this state is that known as "amaurotic family idiocy." In this rare condition, only seen so far in the children of Hebrew parents, there is in association with the symptoms just described a pathognomonic ocular lesion, consisting in the appearance of a whitish-gray patch in the region of the macula lutea, which covers an area nearly twice the size of the optic disc.

FIG. 40.



Spastic diplegia, congenital, presenting choreiform and athetoid movements. (DERCUM.)

In both this and the infantile cerebral form of spastic paraplegia the pyramidal tracts are degenerated.

Acute cerebral paralysis of infancy is to be separated from spinal paralysis of acute or subacute poliomyelitis anterior of infancy, or the rare poliomyelitis of later life. In these there may be bilateral paralysis, although only one leg and foot are usually involved. Like the paralysis from cerebral disease, spinal paralysis comes on suddenly, but there is this marked difference, viz., that in old cerebral paralysis there are spastic rigidity and spastic contractures, no atrophy of the muscles, until by disuse or secondary changes in the cord the muscles lose their nutrition, there is marked increase of the reflexes, and the electrical reactions remain normal for a long period of time; whereas in the early stages of acute spinal paralysis there is an entire absence of spastic contraction, the muscles being peculiarly lax, flabby, and lifeless; contractions with resulting deformity only arising from the action of healthy muscles robbed of their natural antagonists. The contractures of the leg which occur in acute infantile paralysis of spinal origin are not spastic, and are often only developed upon intention-movements or by the unopposed healthy muscles.

Care should be taken that the spastic paraplegia of rickets is not mistaken for a birth-palsy.

Spastic paraplegia in early childhood, when not due to cerebral lesions, as already discussed, is usually due to Pott's disease. The reflexes are exaggerated, the hands are drawn up, and the feet are extended. If the lumbar cord is diseased, the reflexes are lost. Inquiry will perhaps reveal a history that the child has been easily tired before the paralysis came on, and has complained of belly-ache, which has really been due to pain along the intercostal nerves from the irritation at their roots. Thus, pain in the region of the navel suggests inflammation at the eighth dorsal vertebra, or at the ensiform cartilage at the fourth or fifth dorsal vertebra. Early in the disease pressure on the spinal cord may increase the reflexes. The area of the cord involved can be determined by the symptoms as detailed on pages 121 and 122. The prognosis is not always unfavorable, as extraordinary recoveries take place.

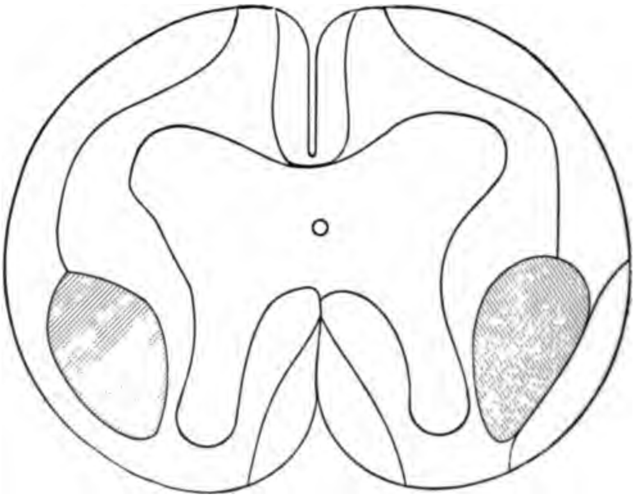
The natural sequence is to pass on to a consideration of the other forms of spastic spinal paraplegia, and to take up first of all its manifestation in children. This occurs in what is known as hereditary spastic paralysis, which is to be separated from infantile cerebral paralysis by the absence of a history of injury to the head at birth, and the absence of convulsions and defective mental development, all of which appear in the cerebral form. This absence of convulsions and defective mental power in this form of spastic paraplegia almost certainly separates it from the cerebral infantile type of paralysis. It is to be separated from the spastic paraplegia of lateral sclerosis by the facts that it occurs in early life, and that there is a history of heredity, or of several members of the family being affected by the disease. There are usually rigidities and contractures, but the bladder and rectum escape the paralysis, and there are no trophic changes. The reflexes are increased. The disease is rare.

A cerebro-spinal cause of spastic paraplegia in adults is multiple cerebro-spinal sclerosis, in which condition the loss of power amounts to a paresis rather than an absolute paralysis. The presence of intention-tremors, exaggerated knee-jerks and ankle-clonus, nystagmus, and vertiginous, epileptiform, or apoplectiform seizures, with staccato speech, and local areas of loss of power elsewhere, associated with the spastic paraplegia, renders the diagnosis easy. (See early part of this chapter.)

In transverse myelitis there is often in the later stages of the malady spastic paraplegia as a result of the irritability of the spinal centres below the seat of the lesion, and this may cause a spastic state of the muscles. In distinction from lateral sclerosis we find in myelitis that there are girdle-pains, involvement of the bladder and rectum, and sensory paralysis.

In the adult, when there is loss of power in the lower limbs with spastic contraction of the muscles when the patient attempts to move them, so that they become rigid, or if before the stage of rigidity develops the gait is spastic and stiff and the reflexes are greatly exaggerated, the disease is generally lateral spinal sclerosis. (Fig. 41.) There is also in lateral spinal sclerosis absence of both sensory disorders and rectal and bladder troubles, but sometimes there are present excessively hasty urination and defecation. The reason why the

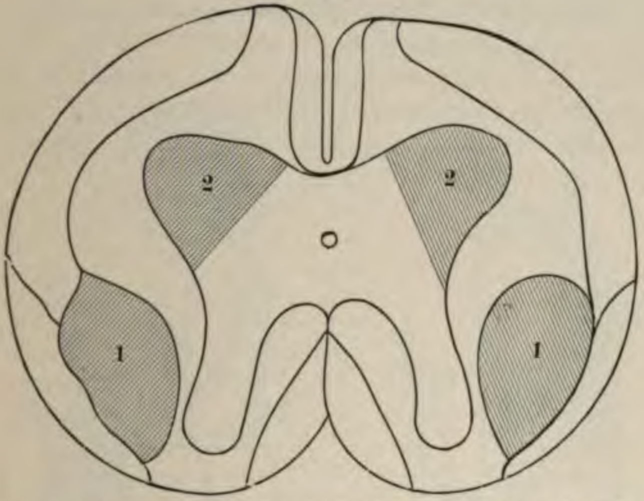
FIG. 41.



Shading shows areas involved in lateral sclerosis, viz., the crossed pyramidal tracts.

reflexes are increased in lateral sclerosis, and similar ailments associated with spastic paraplegia, is that the inhibitory fibres which descend from Setschenow's reflex inhibitory centre in the medulla oblongata are destroyed in the lateral pyramidal tracts. In amyotrophic lateral sclerosis similar symptoms associated with wasting of the muscles are present in the later stages, but in the early stages the arms are chiefly affected by the wasting and paralysis. (Fig. 42.) (See chapter on Hands and Arms.)

FIG. 42.



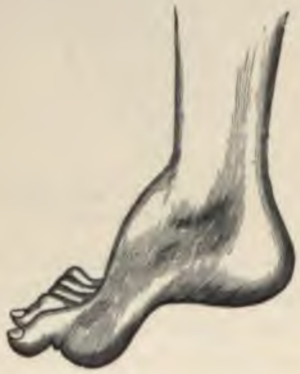
Showing areas of spinal cord involved in amyotrophic lateral sclerosis. 1. Crossed pyramidal tracts. 2. Anterior horns of gray matter containing the trophic cells.

Spastic paraplegia may also be due to spinal pachymeningitis, and the associated symptoms may so closely resemble those of myelitis that a diagnosis is impossible; but the spastic character of the paraplegia, the early appearance and severity of the pain, and the comparatively slow development of the symptoms in pachymeningitis will aid in separating the two affections, as will also the presence of persistently increased reflexes from the first. Sensory disturbances, aside from pain, are common in myelitis, but rare in this condition. If the inflammatory process becomes widespread, there may be sensory disorders and trophic sloughs, owing to invasion of the portions of the cord connected with the sensation and nutrition by a secondary myelitis. The development of signs of spinal caries, sepsis, or a psoas abscess in such cases at once shows the condition to be meningeal in origin, and the history of traumatism will point to meningitis rather than myelitis.

Spastic paraplegia, greatly increased tendon-reflexes, low muscle-tension, vesical disorder, and slight sensory disturbances in an adult should make the physician think of spinal syphilis.

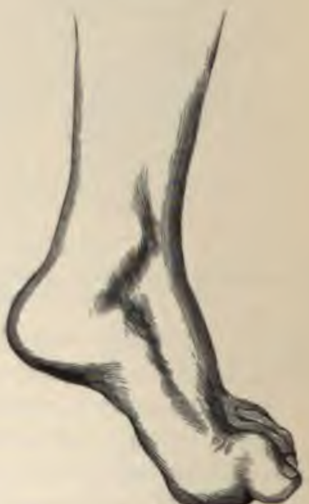
Spastic Monoplegia or Contracture.—The claw-hand spoken of in the chapter on that part of the body is represented by a similar deformity in the foot which may arise from the same causes, in regard to the nervous lesions, and depend upon atrophy of the interossei and other intrinsic muscles of the foot (Fig. 43); but progressive muscular atrophy rarely involves the foot, although it may begin there. When progressive muscular atrophy does begin in the lower extremities, it may fall into the class called progressive neural muscular atrophy, or the peroneal or leg-form of progressive muscular atrophy which affects the leg chiefly and rarely involves the foot proper first. The extensor muscles of the toes lose their power, the interossei waste, the foot may be flattened or claw-shaped, or, in other instances, any one of the forms of club-foot may develop. If the deformity is bilateral, it is a strong evidence of its being the leg-type of progressive muscular atrophy, and not due to infantile paralysis. There will probably be a history

Fig. 43.



"Claw-foot" from atrophy of interossei and other intrinsic muscles of foot. (Duchenne.)

Fig. 44.



Pes equinus in a boy five years of age, from atrophy of tibialis anticus. (Sachs.)

of heredity in such cases. This state of the foot must be carefully separated from the pes equinus seen as a result of progressive and acute infantile spinal paralysis involving the tibialis anticus as seen in Fig. 44. The toes are hyperextended, and the foot is very broad when viewed from side to side at the metatarsal joints. It is stated that this sign is considered characteristic of the early development of the disease in families with the heredity. Sometimes in place of this deformity the foot becomes almost parallel with the tibia in excessive extension, with eversion as the result of shortening of the peroneus longus. (Fig. 45.) In other instances the deformities undergo marked changes as the disease progresses, so that they not only grow worse, but are altered in type. (Figs. 45, 46, 47, and 48.) In distinction from ordinary progressive muscular atrophy this leg-type often has marked disturbance of sensation associated with it. (Dana.) It generally occurs in males. According to Marie, another form of claw-foot is seen in Friedreich's ataxia, there being associated with it club-foot.

FIG. 45.

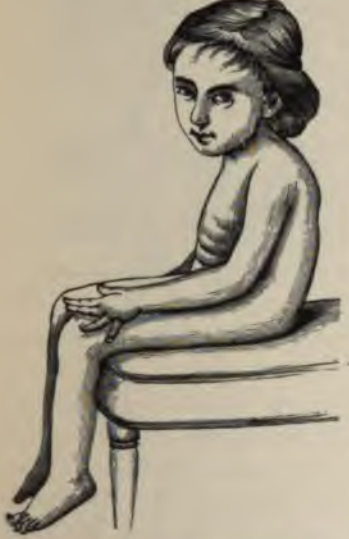
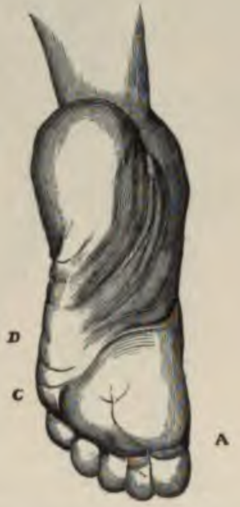


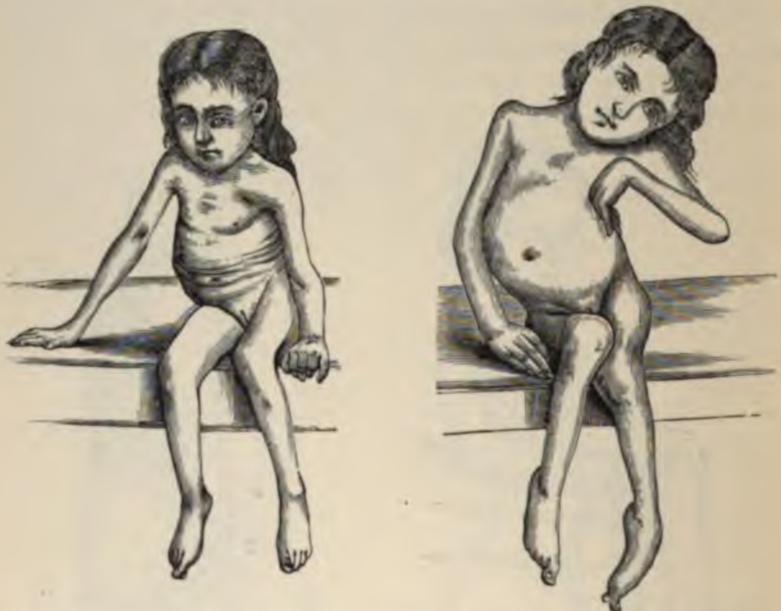
FIG. 46.



Plantar surface exhibiting changes due to contracture of peroneus longus, shortening of transverse diameter, A, C, and torsion of foot, D. (SACHS.)

Progressive neural muscular atrophy is a rare disease, which must be separated from multiple neuritis by the pain of the latter affection and the fact that neuritis rarely produces double club-foot, and, further, that in neuritis there is no history of heredity. From poliomyelitis we separate it by the fact that in this peroneal type of paralysis the onset is more slew and by the fact that there is a loss of the reflexes in severe poliomyelitis, though they are preserved for a long time in the peroneal type. From Friedreich's ataxia it is separated by the fact that in that disease the reflexes are lost, there is a peculiar unsteadiness in walking, and an absence of electrical changes in the muscles.

Fig. 47. Fig. 48.

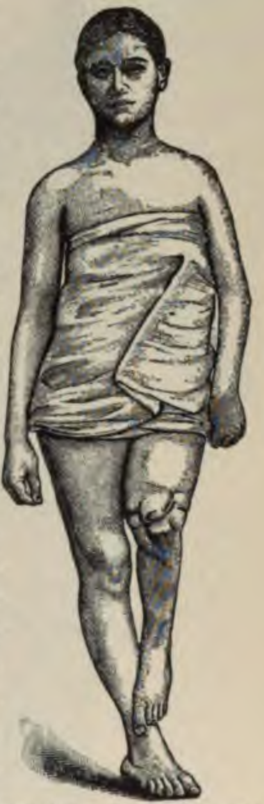


Case of progressive muscular atrophy in a child with a spinal lesion. The four figures (45, 46, 47, and 48) illustrate the progressive wasting of the muscles and the deformities resulting thereform. In Figs. 45 and 48 hyperextension of the fingers and of the big toe is very striking. (Thomson and Bruce.)

A shrivelled, undeveloped foot and leg with drawing up and deformity are seen most commonly as the ultimate result of the acute cerebral paralysis of infancy. (Fig. 49.)

A very important form of contracture following paralysis or occurring without it, which is apt to lead to a mistake in diagnosis, is that seen in hysteria (Figs. 50 and 52). As a rule, the contractures come on in association with paraplegia. Sometimes, however, they affect the arms or an arm. It is a characteristic of these contractures due to hysteria that they set in suddenly, and are always accompanied by such hysterical symptoms as borborygmi, ovarian tenderness, and often areas of anæsthesia. Weir Mitchell has divided these cases into two forms. The first only involves single parts or limited muscle-groups, and, though the contractures may last for years, joint- or muscle-changes do not occur. In the second class, one limb after another is attacked until all means of locomotion, or even moving the trunk, are lost, and the muscles, joints, and areolar tissue undergo organic changes. The reflexes are lost in such cases in the late stages, and the electrical reaction of the muscles is impaired. The diagnosis is to be reached by the sex, the personal history, the history of the illness, the presence of anæsthesia (see chapter on Skin), and hyperæsthesias. Usually the contracture comes on suddenly; it is very rigid, and the muscles on both sides of the limb are fixed—that is, the contracture involves antagonistic muscles. Sleep does not always cause a relaxation of hysterical contraction, but ether or chloroform usually does so. (See chapter on Hands and Arms.)

FIG. 49.



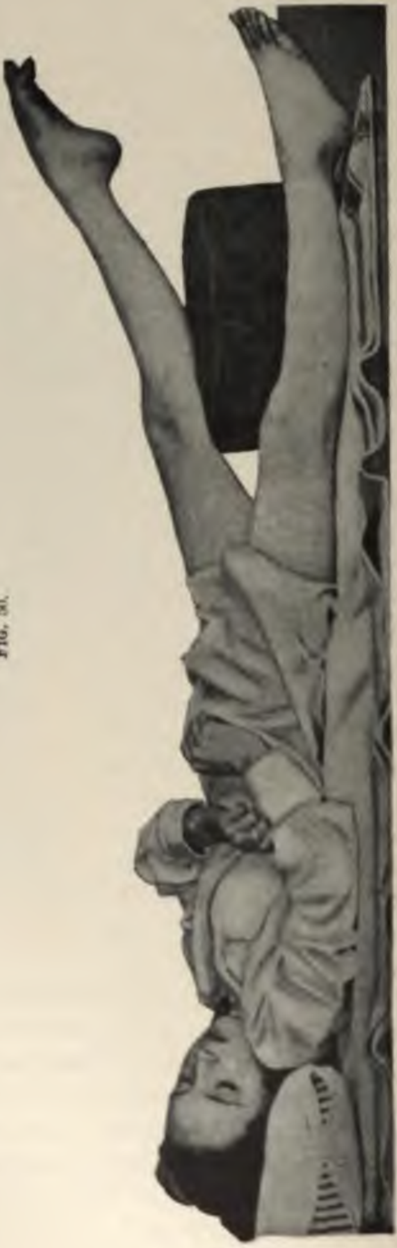
Shrivelled foot of infantile cerebral paralysis. (HIRT.)

Deformity or distortion of the legs may result from the secondary muscular atrophy following upon chronic inflammation in a joint or joints. The muscular wasting under these circumstances may arise from neuritis, which is associated with the arthritis, but its cause is often difficult to discover.

Very rarely universal muscular atrophy follows joint-changes.

NON-SPASTIC PARAPLEGIA. Passing from spastic paraplegia we come to those forms of paraplegia lacking this peculiarity. They are quite numerous and important. If the paraplegia comes on suddenly, the cause may be hemorrhage into the substance of the cord or into the spinal membranes, or be due to compression or

FIG. 50.



Hysterical spasm and contracture, showing the condition of the affected leg in the recumbent position. (BRAMWELL.)

destruction of the cord by injuries of the back, whereby there is laceration of the soft parts or fracture or dislocation of the vertebræ, or be due to acute transverse myelitis.

FIG. 51.

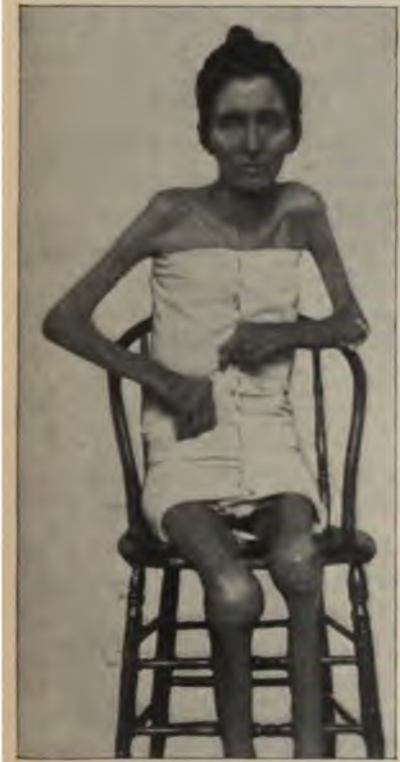


FIG. 52.

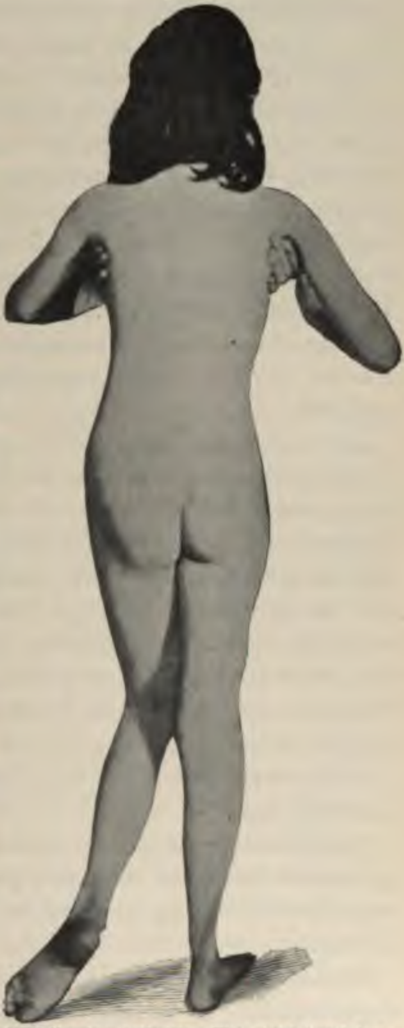


Fig. 51.—A case of universal muscular atrophy following arthritis. The right hand is a "seal-fin" hand. From the author's wards.

Fig. 52.—Hysterical spasm and contracture, showing attitude in erect position. (BRAMWELL.)

When the paraplegia is slower in onset but not sudden, the spinal causes are acute ascending paralysis or Landry's paralysis, acute central myelitis, and acute transverse myelitis. On the other hand, the slowly oncoming non-spastic paraplegias are due to chronic myelitis, to locomotor ataxia, poliomyelitis, neuritis, and pressure due to disease of the vertebræ or to spinal tumors. Finally, we have what are called reflex and hysterical paraplegias.

Hemorrhage into the spinal cord is an exceedingly rare condition unless preceded by grave disease of its tissues. Indeed, the existence of such a condition in man has been denied. The patient, previously in good health, is stricken suddenly to the ground, and there may be almost as much cerebral disturbance as in cerebral apoplexy, but consciousness is generally preserved. The amount of paraplegia may be instantly complete, or not be complete for twenty-four hours. Bedsores speedily develop, and death ensues from exhaustion or from extension of the hemorrhage upward to the vital centres. Practically identical symptoms ensue when the hemorrhage takes place between the membranes covering the cord. In both instances the reflexes are lost if the hemorrhage be sufficient to produce total paralysis.

If, on the other hand, after a prodromal period of short duration, during which there is some fever, the patient is suddenly attacked with paraplegia, the cause may be the acute ascending myelitis of Landry, and the rapid extension to the trunk, the arms, and the respiratory muscles, with the consequent early death of the patient, will confirm the diagnosis. There is usually no involvement of sensation or trophic paralysis, and the sphincters of the bladder and rectum escape the paralysis. Landry's paralysis is very rare. Similar symptoms associated with sensory disturbances are probably due to a polyneuritis.

Diller and Meyer state that the cardinal points for diagnosis of Landry's paralysis are:

1. Flaccid paralysis of the muscles, spreading rapidly from one point over the rest of the body, generally beginning in the legs, but sometimes following the reverse order, as in the French zoölogist Cuvier.
2. Absence of muscular atrophy and of electrical reaction of degeneration.
3. Tendon and superficial reflexes absent.
4. Sensibility not, or only slightly, impaired.
5. Sphincters, as a rule, intact (exceptions rather frequent).

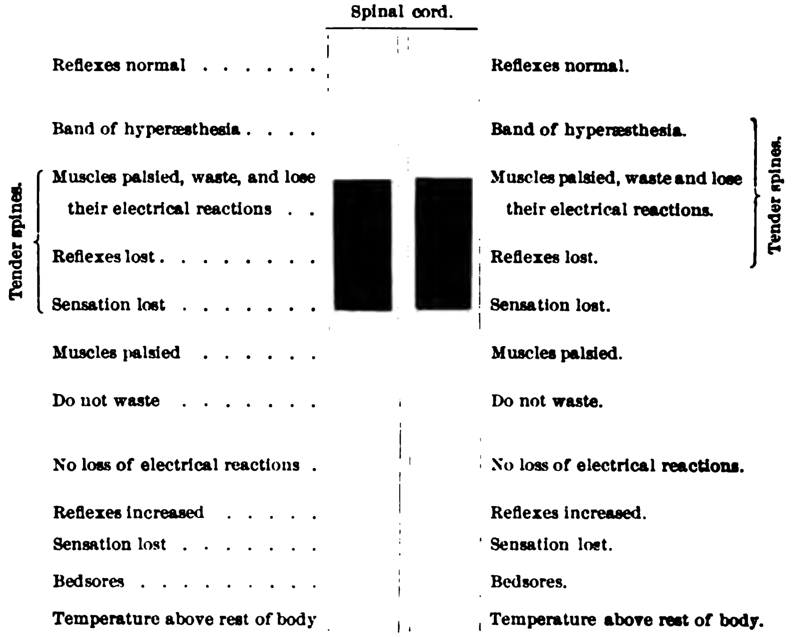
By far the most common cause of paraplegia is myelitis in one of its forms; but, whether the onset be rapid or slow, it must be remembered that the symptoms of myelitis depend, first, upon the level at which the spinal cord is involved, and, second, upon whether the lesion involves the white matter or the gray. If the lesion is an acute central myelitis of the gray matter, it usually produces many of the symptoms about to be detailed under acute transverse myelitis, but the onset is malignant and the areas involved are usually widespread. It is attended by fever of a marked type, though the temperature of the paralyzed parts is below normal, and by early evidences of trophic lesions. Multiple arthritis may come on. The bladder and rectum are paralyzed, and, finally, delirium may develop. The prognosis is unfavorable. Acute central myelitis is to be separated from Landry's paralysis by the facts that in it sensation is lost, and there are rectal and vesical paralysis, fever, and rapid trophic changes. From polyneuritis it is separated by the facts that there are no great trophic changes in this form of neuritis, and the rectum and bladder are rarely paralyzed.

The symptoms of acute transverse myelitis are capable of being divided into three groups, in the first of which the onset is as sudden as is that of apoplexy, in the second the symptoms come on quickly, and in the third more subacutely. In the acute forms, however, the history will be that after a period of numbness, heaviness, and weakness of the legs, with more or less pain the back, the patient has found it impossible to move his legs, has lost control of his bladder and rectum, or suffers from retention of the urine and feces instead, and at the same time has developed anæsthesia of his lower extremities and the girdle-sensation, or, if the lesion be situated high up in the cord, tingling in his arms (see chapter on Skin). The reflexes may be abolished at first, and then return in an exaggerated form in the segments of the cord below the area affected. In other cases the reflexes do not return if the lesion is completely transverse. The patient is speedily bedridden, and to these symptoms just detailed is soon added the development of bedsores and sloughs on dependent parts of the legs or on the buttocks, followed, it may be, by death from exhaustion, although the case may survive for months and even become somewhat better. If improvement takes place, sensation returns in the course of from one to six months, some motion in from six to eighteen months, and, finally, spasms and contractures may result from descending degeneration of the lateral tracts.

The following diagram from Seymour Taylor's *Index of Medicine* shows the effect of a lesion in the spinal cord in transverse myelitis.

SYMPTOMS IN TRANSVERSE MYELITIS.

The darkened portion represents the seat of lesion.



In cases in which paraplegia results from the more subacute form of transverse myelitis the symptoms are usually not quite so rapid in their onset as in the type just named. The patient first notices that his bladder and rectum are unduly irritable, and in his limbs there may be subjective sensory disturbances (see **Paræsthesia** in chapter on the Skin). The motor symptoms begin by a feeling of heaviness or inability to quickly move the lower limbs, so that the patient feels tired on slight exertion. Soon these symptoms deepen into absolute anæsthesia and motor paralysis, and the girdle-sensation on the trunk becomes well developed (see chapter on Skin). The bladder, which at first was irritable, may now be toneless, paralyzed, and retentive or incontinent: retentive if the lesion is above the lumbar cord; incontinent when the lower part of the lumbar enlargement is diseased. The reflexes may at first be abolished, but very soon some of them return, only those reflexes the centres for which are destroyed by the transverse lesion being abolished; that is, the reflexes recover after the first shock of the attack, and those muscles and tendons having spinal centres below the lesion have their reflexes increased because they are cut off from the inhibiting centres higher up in the cord or medulla. The muscles of the legs, which at the first shock of the onset of the malady were all flaccid and paralyzed, now divide themselves into two classes, those that are connected with the diseased part of the cord, which remains paralyzed, and those which are connected with the lower centres, which recover some power; but as the lesion is so placed as to cut them all off from cerebral influences, voluntary motion is lost as completely as if all were deprived of spinal influence. The truly paralyzed muscles waste, but the others which have unimpaired spinal centres do not, except very slowly from disuse. On the contrary, they often become spastically contracted. Other trophic changes, such as bedsores and bullæ, develop in the skin connected with the diseased focus, but not in that connected with centres below the lesion. Anæsthesia is present because the lesion prevents the sensory impulse from reaching the brain (see chapter on Skin). When the entire cord is not evenly involved in the transverse lesion certain groups of muscles partly escape. It is asserted that the extensors escape oftener than the flexors. The height of the paralysis also depends upon the situation of the lesion of the cord, and if high enough to involve the cervical region, and yet not high enough to paralyze the diaphragm and cause death (third or fourth cervical), there may be contraction of the pupil by involvement of the fibres from the nucleus of the third nerve, which runs down the cord to the last cervical vertebræ before joining the sympathetic. When the legs become spastic late in transverse myelitis the cause is supposed to be a descending degeneration in the pyramidal tracts.

The symptoms of chronic transverse myelitis producing paraplegia are practically identical with the more acute form just described, except that they are very slow in their development.

Having discussed the various forms of myelitis, we have still to study the question as to the seat of the lesion in each form. Before doing this, however, it is to be remembered, in studying the relationship of the spinal cord to the vertebræ, that the so-called segments of the cord in no way correspond with the various segments of the spinal column bearing similar names. Thus the cord extends only to the level of the upper part of the second lumbar vertebra, although the spinal canal reaches much lower than this. There are 31 segments of the cord, each of which gives off a pair of spinal nerves. The first segment of the cord is at the foramen magnum and the last at the second lumbar vertebra.

This is well shown in the accompanying figure, modified from one by Gowers. (Fig. 53.)

FIG. 53.

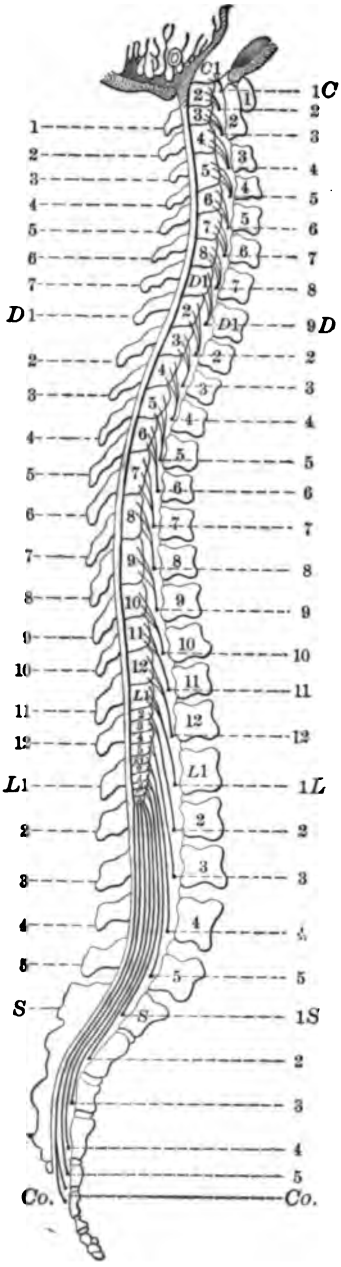


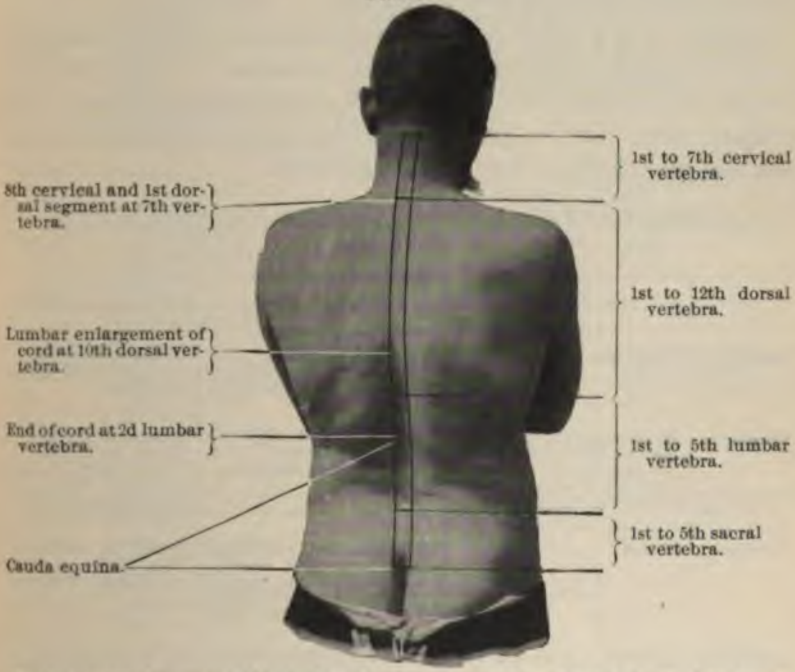
FIG. 53.—Showing relation of segments of cord to vertebræ and relation of spinal nerve-roots to the cord and their levels of exit (modified from Gowers). The figures to the left refer to the spines of the vertebræ, the central figures to the segments of the cord, the right-hand figures to the bodies of the vertebræ.

Not only is this true, but the nerves do not emerge from the spinal canal where they leave the cord, but at a lower level. In the case of the lumbar and sacral segments of the cord the nerves form a bundle which extends down the remaining part of the spinal canal.

There are two areas in the cord of greater importance than the others, namely, the cervical enlargement, which gives off the nerves to the upper extremities, and the lumbar enlargement which supplies the lower extremities. The eighth cervical and first dorsal segments of the cord lie opposite the spine of the seventh vertebra and the lumbar-sacral enlargement opposite that of the spine of the tenth dorsal vertebra.

Let us suppose that a patient presents himself with the following condition: there is complete paralysis of his arms and legs, with paralysis of the muscles of the trunk, and total anæsthesia of the same areas. The legs are in a state of spastic paralysis, their reflexes are increased, and their nutrition is unimpaired; although the arms are found relaxed and flaccid, devoid of reflex excitability, and undergoing degenerative atrophy. The bladder and rectum are not retentive. All these symptoms point to a transverse lesion of the spinal cord in the cervical region, probably between the fifth cervical and first dorsal vertebræ.

FIG. 54.


1st to 7th cervical vertebra.
8th cervical and 1st dorsal segment at 7th vertebra.
1st to 12th dorsal vertebra.
Lumbar enlargement of cord at 10th dorsal vertebra.
End of cord at 2d lumbar vertebra.
1st to 5th lumbar vertebra.
1st to 5th sacral vertebra.
Cauda equina.

Showing the surface-areas of the back corresponding approximately to the areas of the spinal cord supplying the trunk and limbs, according to the diagram on the preceding page.

If, on the other hand, the upper extremities are not affected (except, perhaps, the small muscles of the hand), but there is the same loss of power in the legs, with spastic contraction of the muscles, and the other symptoms just named are present, combined with degeneration of the muscles of the trunk, the lesion is probably somewhere between the second and twelfth dorsal vertebræ.

Again, if the paralysis of motion and sensation be only in the lower limbs, and there be flaccidity of the muscles (where before we discovered spastic contraction), with muscular degeneration, loss of reflexes, and paralysis of the bladder and rectum, the lesion is between the tenth dorsal and first lumbar vertebra.

Still further, if there be loss of power with degeneration of the small muscles of the feet, and loss of sensation of the outside of the feet and toes, and of the skin about the anus, with preservation of power in thighs and of the patellar reflex, the lesion is at the end of the cord in the area of the cone.

In this connection the reader should study that part of the chapter on the skin which deals with anæsthesia.

Finally, it is possible for disease of the cauda equina to produce symptoms of a lumbar-sacral lesion, owing to the fact that this bundle is composed of fibers derived from these two areas. The patellar reflex may be preserved, as the lesion is below the reflex arc, and all the fibres may not be involved.

DIFFERENTIAL DIAGNOSIS OF LUMBAR, DORSAL, AND CERVICAL MYELITIS.[1]

	Lumbar myelitis.	Dorsal myelitis.	Cervical myelitis.
Paralysis.	Paraplegia.	1. Dorsal, abdominal, and intercostal muscles, according to height of lesion. 2. Legs.	Neck-muscles, diaphragm, arms, trunk, and legs.
Sensation.	Pains in legs, or girdle-pains around loins; hyperæsthetic zone around loins; anæsthesia of legs, complete or uneven distribution.	Girdle-pain and hyperæsthetic zone between ensiform cartilage and pubes.	Hyperæsthesia and pains in certain nerve-distributions of arms; below this anæsthesia of arms, body, and legs.
Atrophy.	Of legs.	Of dorsal and abdominal (and intercostal muscles not subject to examination) corresponding to height of lesion; sometimes mild and slow of legs.	Atrophy of neck-muscles (rare) or more commonly of arms.
Electrical reaction.	R. D. in atrophied muscles; or in mild cases quantitative diminution.	R. D. in dorsal and abdominal muscles; slight quantitative changes only in legs when wasted.	R. D. in atrophied muscles.
Bladder.	Incontinence from paralysis of sphincter.	Retention, or intermittent incontinence from reflex action; later from overflow. Cystitis common.	Same as in dorsal myelitis.
Bowels.	Incontinence from paralysis of sphincter; disguised by constipation.	Involuntary evacuation from reflex spasm or constipation.	Same as in dorsal myelitis.
Reflexes, superficial,	Lost.	Temporary loss, then rapid increase.	Same as in dorsal myelitis.
Reflexes, deep.	Lost.	Temporary loss, then slow increase.	Same as in dorsal myelitis.
Priapism.	Absent.	Often present.	Often present.

[1] From Prince's article in Dercum's Nervous Diseases.

This subject is still further subdivided and elucidated by the following table and by the illustration given on page 119.

LOCALIZATION OF THE FUNCTIONS OF THE SEGMENTS OF THE SPINAL CORD. (According to STARR.)

Segment.	Muscles.	Reflex.	Sensation.
II. and III. Cervical.	Sterno-mastoid. Trapezius. Scaleni and neck. Diaphragm.	Hypochondrium. (?) Sudden inspiration produced by sudden pressure beneath the lower border of ribs.	Back of head to vertex. Neck.
IV. Cervical.	Diaphragm. Deltoid. Biceps. Coraco-brachialis. Supinator longus. Rhomboid. Supra- and infra-spinatus.	Pupil. 4th to 7th cervical. Dilatation of the pupil produced by irritation of the neck.	Neck. Upper shoulder. Outer arm.
V. Cervical.	Deltoid. Biceps. Coraco-brachialis. Brachialis anticus. Supinator longus. Supinator brevis. Rhomboid. Teres minor. Pectoralis (clavicular part). Serratus magnus.	Scapular. 5th cervical to 1st dorsal. Irritation of skin over the scapula produces contraction of the scapular muscles. Supinator longus. Tapping its tendon in wrist produces flexion of forearm.	Back of shoulder and arm. Outer side of arm and forearm, front and back.
VI. Cervical.	Biceps. Brachialis anticus. Pectoralis (clavicular part). Serratus magnus. Triceps. Extensors of wrist and fingers. Pronators.	Triceps. 5th to 6th cervical. Tapping elbow tendon produces extension of forearm. Posterior wrist. 6th to 8th cervical. Tapping tendons causes extension of hand.	Outer side of forearm, front and back. Outer half of hand.
VII. Cervical.	Triceps (long head). Extensors of wrist and fingers. Pronators of wrist. Flexors of wrist. Subscapular. Pectoralis (costal part). Latissimus dorsi. Teres major.	Anterior wrist. 7th to 8th cervical. Tapping anterior tendons causes flexion of wrist. Palmar. 7th cervical to 1st dorsal. Stroking palm causes closure of fingers.	Inner side and back of arm and forearm. Radial half of the hand.
VIII. Cervical.	Flexors of wrist and fingers. Intrinsic muscles of hand.		Forearm and hand, inner half.
I. Dorsal.	Extensors of thumb. Intrinsic hand muscles. Thenar and hypothenar eminences.		Forearm, inner half. Ulnar distribution to hand.
II. to XII. Dorsal.	Muscles of back and abdomen. Erectores spinæ.	Epigastric. 4th to 7th dorsal. Tickling mammary region causes retraction of the epigastrium. Abdominal. 7th to 11th dorsal. Stroking side of abdomen causes retraction of belly.	Skin of chest and abdomen, in bands running around and downward corresponding to spinal nerves. Upper gluteal region.
I. Lumbar.	Ilio-psoas. Sartorius. Muscles of abdomen.	Cremasteric. 2d to 3d lumbar. Stroking inner thigh causes retraction of scrotum.	Skin over groin and front of scrotum.
II. Lumbar.	Ilio-psoas (sartorius). Flexors of knee (Remak). Quadriceps femoris.	Patellar tendon. Striking tendon causes extension of leg.	Outer side of thigh.

Segment.	Muscles.	Reflex.	Sensation.
III. Lumbar.	Quadriceps femoris. Inner rotators of thigh. Abductors of thigh.		Front and inner side of thigh.
IV. Lumbar.	Abductors of thigh. Adductors of thigh. Flexors of knee (Ferrier). Tibialis anticus.	Gluteal. 4th to 5th lumbar. Stroking buttock causes dimpling in fold of buttock.	Inner side of thigh and leg to ankle. Inner side of foot.
V. Lumbar.	Outward rotators of thigh. Flexors of knee (Ferrier). Flexors of ankle. Extensors of toes.		Back of thigh, back of leg, and outer part of foot.
I. to II. Sacral.	Flexors of ankle. Long flexor of toes. Peronei. Intrinsic muscles of foot.	Plantar. Tickling sole of foot causes flexion of toes and retraction of leg.	Back of thigh. Leg and foot, outer side.
III. to V. Sacral.	Perineal muscles.	Foot-reflex. Achilles tendon. Overextension of foot causes rapid flexion; ankle-clonus. Bladder and rectal centres.	Skin over sacrum. Anus. Perineum. Genitals.

Paraplegia when due to locomotor ataxia is nearly always so surrounded by other typical symptoms of this disease as to render its separation from the paraplegia of myelitis easy, and, further, it is rarely a true loss of power. The stabbing and darting pains of ataxia (see chapter on Pain), the presence of the Argyll-Robertson pupil, the absence of the patellar reflex, and the atrophy of the optic nerve are all characteristic of ataxia, and absent in myelitis (see also early part of this chapter on Gait).

The symptoms of lateral sclerosis and amyotrophic lateral sclerosis have already been discussed under Gait and Spastic Paraplegia, but in the paraplegia called "ataxic paraplegia," also already discussed, there are in association lateral sclerosis and posterior sclerosis, and for this reason some of the symptoms of both are found to be present. Thus, in addition to loss of power there is a spastic condition of the legs with exaggerated reflexes, absence of the Argyll-Robertson pupil and of crises of pain, but the Romberg symptom, or swaying when the eyes are closed, is present. The condition which most closely resembles ataxic paraplegia is that of tumor of the middle lobe of the cerebellum, but in such cases we have, in addition, headache, vertigo, optic neuritis, titubation, and sometimes vomiting.

The onset of the paraplegia in a young child, preceded by an attack of fever, vomiting, restlessness, and general illness, lasting but a few hours or days, and which may be complicated by convulsions, all point to the cause being poliomyelitis of a severe type. The legs are, however, as a rule, completely paralyzed for but a brief period after the attack. Eventually the storm clears off, and only the muscles directly connected with the diseased cells in the cord (anterior cornua) remain paralyzed. There is no loss of sensation, but reflex action is abolished in the paralyzed parts. Far and away the most important point in the diagnosis is the symptom of rapid wasting of the muscles in the paralyzed parts and the rapid development of coldness in these areas, which is due to the destruction of the trophic centres in the spinal cord (see Fig. 55).

Paraplegia resulting from tumor of the cord or its membranes only ensues when the growth is so placed as to cut off all the motor tracts supplying both limbs, which is rarely accomplished until after a long history of more or less well-developed motor and sensory failure. The paralysis is developed in the areas supplied by the centres in the cord below or at the level of the growth, and the violent pain nearly always present in cases of tumor points to the diagnosis. Very painful paraplegia, therefore, indicates spinal tumor as its cause. The area of anæsthesia and the muscles involved may also give definite information as to the seat of the growth (see chapter on the Skin, and Starr's table just quoted).

If the paraplegia be due to compression from fracture or dislocation of the vertebræ or to other direct injury, the history of the patient and the evidences of external local mischief will decide the diagnosis.

Sometimes during the course of severe disease, producing irritation of the bladder, kidney, bowels, or rectum, as in violent cystitis, stone in the kidney, and dysentery, paraplegia comes on, due in some cases to an infectious myelitis, but in others to what is apparently only a reflex paralysis, as it often passes away with the removal of the source of irritation. Even worms in the intestine have produced such a paralysis, and their removal has been followed by cure. Generally sensation in the limbs is unimpaired and the bladder and rectum act normally. Sometimes, however, in the presence of severe renal disease, as renal calculus, there may be all sorts of disturbance of sensation and pain, as well as great motor paralysis, with total loss of reflexes, following an exaggeration of the reflexes. Probably these severe cases are always due to a coincident myelitis rather than to a reflex irritative cause.

No form of paraplegia presents so many types or represents so many organic diseases as does that due to hysteria, for there may be not only great loss of motion, but exaggerated or lost reflexes, relaxation or spastic contraction of the muscles, anæsthesia and hyperæsthesia, pain or no pain. The very occurrence of such irregular manifestations in a young, neurotic girl, the facts that the anæsthetic areas constantly tend to shift their position, and, finally, that the contractures, if present from hysteria, disappear on administering an anæsthetic to a stage in which muscular relaxation is produced in the ordinary individual, aid us in making what is, in some cases almost impossible, a diagnosis (see that part of this chapter on Contractures).

A pseudo-paralysis of the legs with immobility sometimes occurs as a symptom of scorbutus in infancy. The parents notice that the child flinches when it is picked up or handled, and seems as if tender from rheumatism. Often the gums are swollen and bleeding, and purpuric eruptions appear on the skin. The shafts of the bones of the legs or of the arms may be enlarged, and hæmaturia or bloody stools may appear.

Pseudo-paraplegia may also occur in rickety children from faulty muscular and bony development. It is to be separated from the ordinary paraplegias of childhood by the state of the bones, the presence of knee-jerks, and the absence of local wasting or spasm although general spasms, or carpo-pedal spasm, is often seen in rickety children.

Not uncommonly a partial paraplegia occurs as a result or sequel of diphtheria. The condition, however, is more ataxic than paraplegic, and Bourges asserts that there is no muscular atrophy such as occurs in true paraplegia due to neuritis, or in that due to some spinal lesions.

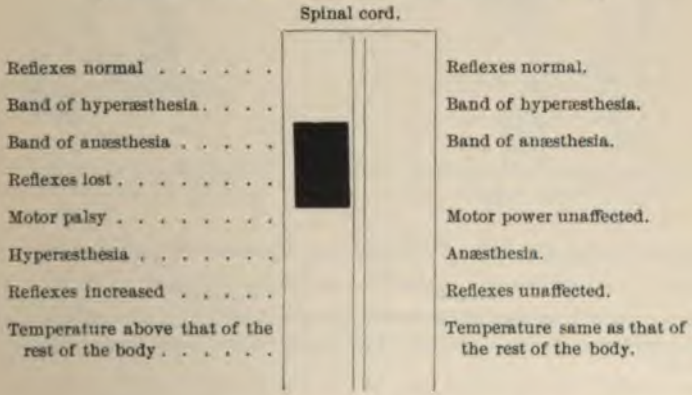
When neuritis produces sudden paraplegia the symptoms very closely resemble those of acute myelitis. Neuritis may also cause pseudo-tabes if its onset is slow. The neuritis is always multiple and involves the arms and the body after affecting the legs; there is well-developed anæsthesia (see chapter on Skin), preceded by sensory disturbances and marked muscular and nerve-trunk tenderness; but there is no girdle-sensation, as there is in myelitis and tabes. There are often trophic changes in the skin in neuritis (see chapter on Skin), but no bedsores as in myelitis. J. M. Da Costa stated that malarial neuritis may cause paraplegia of the lower limbs, but, as a rule, toxic neuritis produces loss of power in the arms. Very rarely paraplegia of the lower extremities results from diabetes mellitus, the lesion being in all probability a multiple neuritis.

A very rare condition, involving not only the legs, but the entire body, is what is called "family periodic paralysis," in which flaccid motor paralysis, with loss of electrical reaction and reflex activity, comes on suddenly, lasts for a few hours or days, and is then followed by perfect health. It is always hereditary.

Monoplegia of a lower extremity may be due to a cerebral lesion or to spinal or nerve-trunk lesions. The cerebral lesion producing monoplegia in one leg is very rare, and if it occurs, at any age, indicates a lesion in the convolutions at the upper end of the fissure of Rolando, and the continuation of this area in the paracental lobule of the marginal convolution. Unlike the paraplegias of infantile cerebral paralysis, monoplegia of the leg very rarely arises from this cause. If there are no signs of cerebral trouble, the presence of a complete leg monoplegia can mean one of several things, namely, a lesion limited to one side of the cord, as, for example, a hemilateral myelitis, hysterical paralysis, in which there will be irregular anæsthesia (see Skin), and the other hysterical signs, or a tumor pressing on the crural nerve in the pelvis, or section of the nerve by injury. Apparent monoplegia may, however, be due to muscular pain or a painful phlebitis producing muscular fixation.

DIAGRAM SHOWING SYMPTOMS IN HEMILATERAL MYELITIS.[1]

(The darkened mass represents the site of the lesion.)



If the condition is due to a lesion on one side of the cord, the symptoms are quite characteristic. There is paralysis of all the muscles of the leg which are supplied by the part of the cord affected or below it. The muscles, the nerve-supply of which comes directly from the affected part, eventually waste and undergo degenerative changes. The most typical symptom of this lesion is, however, the crossed character of the sensory paralysis. That is to say, there is loss of sensation in the limb opposite that in which motion is lost, and in the limb in which motion is lost there is hyperæsthesia, so that the lightest touch may be very painful. The cause of this is obscure, for the studies of Mott have proved that the sensory tracts in the cord do not decussate on entering it, as has been supposed heretofore. There is, however, a symmetrical band of anæsthesia round the body at the level of the lesions, and a similar band of hyperæsthesia above the lesions. The reflexes of the parts supplied by the diseased area are lost, but those supplied by the area below the lesions are increased as in ordinary myelitis. Very commonly the paralyzed limb is over-warm from vasomotor palsy.

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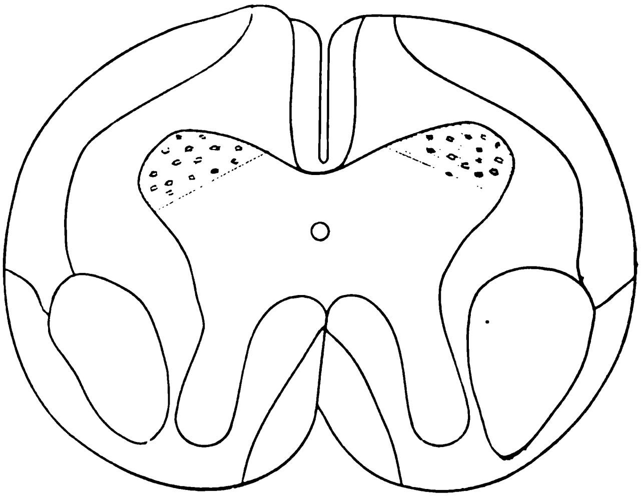
[1] From Seymour Taylor's *Index of Medicine.*

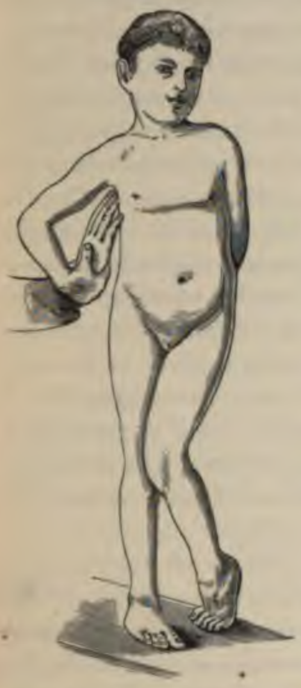
FIG. 55.



Areas involved in acute and chronic poliomyelitis. In children it is sometimes called acute infantile paralysis. Shading shows area of cells in anterior cornua of gray matter which are involved.

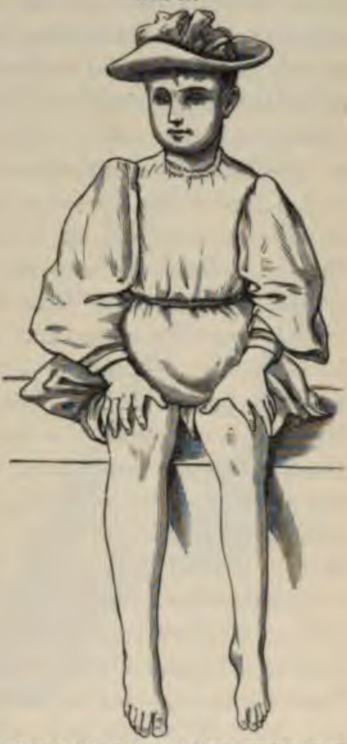
Paralysis of certain groups of muscles or a single muscle in the legs is most commonly due to anterior poliomyelitis or neuritis. (Fig. 55.) In poliomyelitis the child will be found to have loss of power in certain muscles in one or both legs (see also Paraplegia), so that there is a dragging of the toe, or "foot-drop," the shoe becomes irregularly worn through, being dragged on one edge along the ground, the involved muscles being peculiarly relaxed and flaccid, so that the leg may wabble, to use a crude term. This is sometimes called a "Punchinello leg." There is no tendency to spastic contraction, the reflexes are rapidly lost in the affected part, and the muscles speedily waste and develop the reaction of degeneration. (Fig. 57.) When contractures take place they are not spastic, and are due to healthy muscles being unopposed by the diseased ones. The temperature of the paralyzed part is lower than normal. The history in poliomyelitis is that of sudden onset, with fever, vomiting, and restlessness. The two conditions of acute cerebral paralysis and anterior poliomyelitis are so clearly separated in well-marked cases that no error can be made, particularly if the history of the attack be borne in mind, unless it be in the obscure forms

FIG. 56.



Case of acute infantile cerebral palsy for comparison with Fig. 57. (SACHS.)

FIG. 57.



Case of infantile spinal palsy: paralysis and atrophy of left leg chiefly. (SACHS.)

of cerebral infantile palsy in the early stages. The above figures show the two different types of paralysis resulting from these cerebral and spinal diseases in the child. (Figs. 56 and 57.) In acute infantile paralysis of spinal origin the right lower extremity is most frequently affected, after this, a close second, the left leg. Sometimes muscular atrophy may be masked in young children by the abundance of subcutaneous fat. A point of some importance in examining the reflexes is that presence of knee-jerk should not exclude the diagnosis of poliomyelitis, because the reflex act is only destroyed if the centres which cause this jerk are diseased—that is, if the disease has only affected that part of the cord supplying the foot, a tap on the knee may readily produce a response, whereas if the disease be higher up in the cord the reflex will be lost. The chronic anterior poliomyelitis of adult life presents very similar symptoms to the acute form of infancy, but is a very rare disease.

Care must be taken that paralysis of the leg resulting from an injury to the peroneal nerve with resulting neuritis is not mistaken for acute poliomyelitis. The history of an accident, of pain, swelling, and the presence of a bruise aid us in making a diagnosis. If these symptoms occur in an adult, a possible cause is paralysis of the peroneal nerve occurring in the course of tabes. (In connection with this chapter see that on the significance of anæsthesia of the skin.)

Deformities of the Feet and Legs. Much of what has been said in the preceding chapter as to the diseases which produce alterations in the shape of the hand and arm applies equally to the changes from the normal seen in the appearance and movements of the feet and legs. The feet are greatly enlarged symmetrically in acromegaly and in Marie's pulmonary osteo-arthropathy. In the latter disease the enlargement is particularly noticeable because it is the extremities which are chiefly hypertrophied, whereas in acromegaly there is simultaneous enlargement of the shafts of the long bones. (See chapter on Hands and Arms.) It is to be remembered that in both acromegaly and pulmonary osteo-arthropathy the enlargement seems to be due to hypertrophy of all the tissues composing the foot, whereas, on the other hand, in myxœdema the foot, though enlarged, is puffed and swollen in appearance through hypertrophy of the subcutaneous tissues alone. Often the foot appears to be a good deal enlarged as the result of deformity, particularly that which consists in partial displacement of the articular surfaces of the metatarsal and phalangeal bones through the wearing of badly fitting shoes, or joint-troubles, of which we shall speak later.

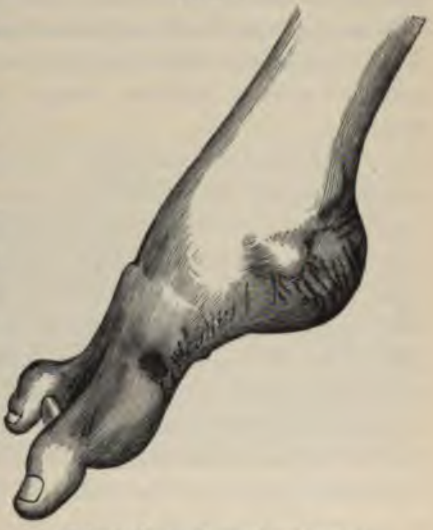
Under the name of "sciopedy" Power has reported a case of congenital symmetrical enlargement of the anterior part of the foot not involving the heel. Any enlargement of the legs associated with this condition, he states, only results from hypertrophy of the muscles resulting from the effort to lift the feet.

In some cases of locomotor ataxia flat-foot from loss of the plantar arch is seen, and various dystrophies of the joints take place as the disease progresses.

The peroneal leg-type of progressive muscular atrophy may so closely resemble the so-called Aran-Duchenne type of progressive muscular atrophy as to defy diagnosis, but, as a rule, the latter disease affects the arms first, and sensation is not involved. (See chapter on Hands and Arms.)

Wasting of the muscles of the inner surface of the foot affecting the big toe, and those on the outer side involving the movements of the little toe, the interossei and the flexor brevis communis, may occur from the neuritis due to locomotor ataxia, and as the plantar aponeurosis retreats from the toes are rendered immovably flexed; in other cases in case of flexion there is strong extension, as in this figure. (Fig. 58.)

FIG. 58.



Tabetic foot. (SHINGLETON SMITH.)

The Joints. The joints of the lower limbs may be swollen from an arthritis arising from many causes, such as septicæmia, gonorrhœal infection, syphilis, acute articular rheumatism, typhoid fever, tuberculosis, locomotor ataxia, hemiplegia, rheumatoid arthritis (arthritis deformans), acute myelitis, cerebro-spinal meningitis, Morvan's disease.

When arthritis is due to gonorrhœal infection it is generally seen in the knees or ankles, and occurs in men as a rule. It is an infectious arthritis and lasts very persistently, often attacking at the same time joints so rarely involved by rheumatism as the jaw, the vertebral joints, and the sterno-clavicular articulation. According to the late Dr. Howard, of Montreal, it occurs in five forms:

a. Arthralgic, in which there are wandering pains about the joints, without redness or swelling. These persist for a long time.

b. Rheumatic, in which several joints become affected, just as in subacute articular rheumatism. The fever is slight; the local inflammation may fix itself in one joint, but more commonly several become swollen and tender. In this form cerebral and cardiac complications may occur.

c. Acute gonorrhœal arthritis, in which a single articulation becomes suddenly involved. The pain is severe, the swelling extensive and due chiefly to periarticular œdema. The general fever is not at all proportionate to the intensity of the local signs. The affection usually resolves, though suppuration occasionally supervenes.

d. Chronic hydrarthrosis. This is usually monarticular, and is particularly apt to involve the knee. It comes on often without pain, redness, or swelling. Formation of pus is rare. It occurred only twice in ninety-six cases tabulated by Nolen.

e. Bursal and synovial form. This attacks chiefly the tendons and their sheaths, and the bursæ and the periosteum. The articulations may not be affected. The bursæ of the patella, the olecranon, and the tendo Achillis are most apt to be involved.

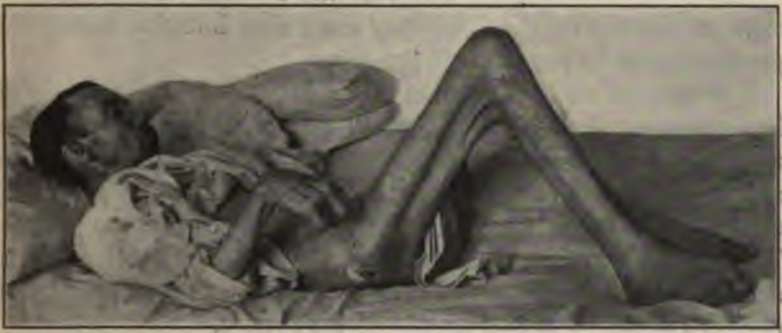
Acute articular rheumatism in the knee or ankle produces swelling of the joint, redness, heat, exquisite tenderness, immobility from pain, swelling of the surrounding tissues. It does not remain for a long period unchanged in one joint, and is a process accompanied by fever.

Acute synovitis is generally the result of an injury, is confined to one joint, is often accompanied by far greater effusion into the joint than is seen in rheumatism, and there is no systemic disturbance. Should a single joint be affected by an aberrant attack of acute rheumatism or synovitis not following injury, the physician should never forget the possibility of its being a syphilitic arthritis.

The most marked alterations in the joints are those produced by advanced locomotor ataxia, and are called arthropathies. Often they are associated with spontaneous fractures of the bones. The knees are most commonly involved, then the ankles and hips. A joint or several joints may become suddenly swollen with or without pain, and without apparent cause until the swelling becomes quite massive. There are then developed osseous hyperplasia and a tendency to dislocation with crepitation on movement, and the ends of the bone become worn away and absorbed. Dislocation and fractures are common, and the bones are atrophied.

In rheumatoid arthritis there is a gradual enlargement of the joints from accumulation of fluid, which in turn is absorbed, leaving the articulating surfaces roughened, uneven, and deformed, but there are no deposits of urate of sodium as in gout, the deformities being due to alterations in the articulating surfaces themselves, and the periarticular development of bone. The disease always remains in the joint originally attacked, although new joints are involved. Pain is often severe, dislocations and fractures are rare, and the small joints are often involved. (Fig. 59.) (See chapter on Hands and Arms.)

FIG. 59.



Atrophy of muscles, fixation of joints, swelling of feet, and bed-sores in case of rheumatoid arthritis; also fixed wrist and neck. (From the author's wards.)

Rheumatoid arthritis when it progresses to an advanced stage causes great deformity by the locking of the joints through the development of osteophytes. By the destruction of the cartilages, wasting of the muscles, and thickening or contraction of the ligaments, it may cause dislocation of all sorts, and false positions. In the great majority of cases it occurs in women between twenty and thirty years of age, but it may develop in early childhood. Pain is severe in some cases, absent in others. The thighs become flexed upon the abdomen, and the leg on the thigh. The number of joints involved varies greatly, but the involvement is generally symmetrical.

Sometimes this disease, which is generally gradual in its onset, becomes very acute, speedily involving many joints, causing swelling of the synovial sheaths and bursæ, and being accompanied by some febrile movement. The suddenness of its onset, the febrile movement when the onset is sudden, and the pain may cause it to resemble acute articular rheumatism, but the absence of redness in the joints and of the migration of the swelling from one joint to another aids in the differentiation. The arthritis of acute central myelitis is sudden in its onset, generally multiple, and accompanied by the other symptoms of that disease (see Paraplegia and Anæsthesia of the Skin.)

The arthritis of cerebro-spinal meningitis is really to be classed as an infectious arthritis, and the presence of the characteristic signs of the disease renders its cause evident. The joints are many of them affected simultaneously with swelling, pain, and serous or purulent effusions. In cases of septic arthritis the joints become swollen and often suppurate, so that the articular surfaces become more or less destroyed. This may occur after infection during the puerperium or in any case of pyæmia.

The onset of an inflammation in the lower end of the femur or in the upper end of the tibia, producing what, at first glance, seems to be an arthritis and sometimes simultaneously involving other areas near joints, should raise a suspicion of acute osteomyelitis, which is a fatal disease in many cases unless surgical aid comes to the patient. The symptoms consist of boring pain in the part, great tenderness and swelling, and the skin soon breaks down as a purulent and offensive discharge makes its way to the surface.

Closely allied to this is the acute epiphysitis of infancy, in which there is suddenly developed a chill followed by great pain and swelling of the joints or their neighborhood. The skin becomes engorged with blood and the joint fills with pus. Care must be taken to separate this condition from rheumatism and the joint-swelling sometimes seen after typhoid fever and that form which is due to infantile scurvy. This state is practically identical with the acute arthritis of childhood.

Great swelling of the thigh or leg occurring in a child may be due to subperiosteal hæmatoma (Möller-Barlow's disease). Aspiration of the swelling will reveal the character of its contents, and the child will usually be a sufferer from rickets.

Although gout is capable of causing deformity in the lower extremities, it has one fact about it which is of practical importance, namely, that it involves the small joints of the foot, while rheumatism attacks the large joints, such as the knee, by preference. Gout involves the feet most commonly, while rheumatism is more frequently seen in the hand, if small joints are affected, and the big toe is the favorite place for gouty manifestation. Aside from the swelling, redness, and exquisite tenderness of gouty joints, all of which symptoms exceed in acuteness, if possible, similar manifestations in acute rheumatism, there is often an additional and permanent cause of deformity in the chalk-stones which are deposited about the joints, and which are never seen in rheumatism. The history of frequently recurring attacks lasting but a few days, accompanied by enlargement of the veins about the joint and shedding of the skin locally, points, when added to the symptoms named, to a typical case of gout. It may be almost impossible to determine whether a case be one of chronic rheumatism or gout unless chalk-deposits can be found. (See chapter on Hand.)

Sometimes in chronic lead-poisoning we have developed what is known as plumbic gout, owing to the deposition of urate of lead and sodium.

The onset of a multiple arthritis, with which there are headache, chills, intense aching in the bones, joints, and muscles, and a fever rising as high as 106° or 107°, and rarely an erythematous rash, may indicate the presence of dengue. The joints are swollen and painful, and often both the large and small ones are involved. Another arthritis, probably infectious, is sometimes seen in epidemic dysentery and in scarlet fever. Rarely immediately after or some months after typhoid fever a hypertrophic osteo-arthritis comes on as a result of a local difficulty produced by the bacillus of Eberth. This is to be separated from ordinary septic arthritis following typhoid fever.

In Schönlein's disease, which is a form of very severe purpura, multiple arthritis, with great pain, and purpuric eruptions occur, and the presence of the subcutaneous exudate with œdema and sloughing of the mucous membrane of the mouth adds to the picture. The patient seems very ill, but death rarely follows. Such cases are rare, but the writer saw one in consultation with Dr. Wilson, of Woodbury, New Jersey, in which alarming sloughs of the tonsils and buccal mucous membrane occurred. (See chapter on Skin.)

Very nearly allied to this are the joint-involvements of hæmophilia, which in their sudden onset and pain closely resemble rheumatism, particularly as the large joints are commonly involved. The history of the patient being a bleeder, or of his being related to one, may clear up the diagnosis.

So-called "spontaneous dislocations of the hip" have been recorded during convalescence from typhoid fever, scarlet fever, and acute rheumatism. The luxation in the latter disease usually follows severe pain, and the ligaments are often found torn from their attachments. In typhoid and scarlet fevers the dislocation occurs insidiously and announces itself by the pain it causes.

Intense swelling of the leg, aside from that due to ordinary œdema, may be due to milk-leg, which is a condition of swelling of the entire limb, generally limited to one side, and seen during the puerperium or after any one of the infectious fevers, such as typhoid. The joints are not particularly affected. On the contrary, the calf of the leg is the part most affected, it being white, firm, hot, but slightly, if at all, œdematous. Pain is excessive, there is entire loss of power in the affected limb, and its temperature is much higher than normal.

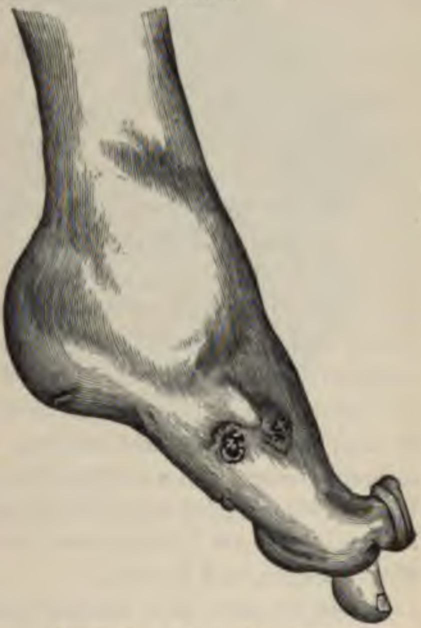
If the swelling of the leg is bilateral and pits on pressure, it is practically always the result of anasarca from renal or cardiac disease; but if unilateral, it may be, as just stated, due to thrombosis of the femoral vein (see chapter on the Skin; Œdema).

Three very important serious alterations in the nutrition of the foot remain to be noted, namely, perforating ulcer due to tabes dorsalis, diabetic gangrene, and senile gangrene. Perforating ulcer usually appears in one foot, beginning with the formation of a bleb, which changes to an abscess, which in turn is followed by necrosis of all the tissues of the foot immediately underlying the destroyed skin. With it are associated the signs of ataxia. Sometimes perforating ulcer of the foot occurs during the course of diabetes mellitus, but it is probable in many such cases that locomotor ataxia is associated with diabetes. (Fig. 60.) In diabetic gangrene the toes are nearly always affected in preference to other parts of the body. An analysis of the urine will aid the diagnosis (see chapter on Skin). In senile gangrene the age of the patient, bad bloodvessels, and the absence of a sufficient cause for gangrene, as for example, trauma, separate the case from any other condition, while the facts that senile gangrene generally affects the inner side of the foot, especially the big toe, and is a dry gangrene, render the diagnosis easy.

Gangrene of the extremities sometimes follows the infectious diseases, such as scarlet and typhoid fevers,[1] from thrombosis of the femoral artery. It may also occur in the course of exophthalmic goitre.

More rarely gangrene of the foot and hand follows embolism due to cardiac valvular disease. It is extremely painful, and septic fever may ensue.

FIG. 60.



Tabetic ulcer. (SHINGLETON SMITH.)

In this connection mention may be made of "Madura foot," or mycetoma, a chronic local disease of tropical climates, and called "fungus foot disease" in India. A small tumor develops on the foot or hand, which, after the lapse of twelve to twenty-four months, bursts and leaves several sinuses from which escape black particles or whitish-red bodies like fish-roe. The disease may spread all the way up the leg. The pale particles in the discharge look like actinomyces.

Ainhum is a disease peculiar to dark-skinned races, characterized by gradual drying up and separation of the toes (by a constriction), usually the little toe. It has been thought to be related to leprosy, but this is doubtful.

Alterations in the appearance of the tibiæ or shins often give us a clear idea of the presence of late syphilis, either because of gummatous swellings in this neighborhood or owing to the development of periosteal thickening and exostoses.

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[1] See "The Medical Complications and Sequelæ of Typhoid Fever," by the author. Lea Brothers & Co., Philadelphia, 1899.

CHAPTER IV.

HEMIPLEGIA.

HAVING considered the manifestations of disease as seen in the arms and legs in connection with monoplegia and paraplegia, spasm and contracture, we must now study the diagnostic meaning of hemiplegia, or that form of paralysis which involves the arm and leg and head on one side of the body. This form of paralysis, when complete, is always due to a lesion arising above the spinal cord—that is, it is cortical, or due to lesions in the lower tracts of the brain; and the character of the paralysis, the association of other symptoms with it, and the history of the patient and his illness will render a diagnosis easy as to the approximate site of the lesion in most cases. The most common causes are hemorrhage into the cerebral tissues from a ruptured bloodvessel, or embolism or thrombosis of some vessel supplying important areas. Still other causes of hemiplegia are brain-tumors, meningeal hemorrhage, degenerative processes, and hysteria.

Before we enter into consideration of the various symptoms resulting from central nervous lesions it is well to stop for a moment for the purpose of clearly understanding the anatomy and physiology of the parts involved, in order that we can properly study the results of lesions in the nerve-centres or nervous tracts.

It is not necessary to remind the reader that the brain is divided into three areas, the frontal area being concerned with intellection, the middle area with motion, and the posterior area with sensation and special sense. These areas are again divided into subareas, each of which governs or is connected with several functions, and still further subdivisions exist, in which reside the centres governing small areas, as, for example, a single muscle or group of muscles. (Figs. 1 and 2, Plate II.) Disease of any part of the brain surface, therefore, modifies more or less the function of that part and the part of the body tributary to it. Beneath the surface, through the so-called white matter, various fibres pass, which carry to or from the centres in the cerebral cortex the impulses connected with their

PLATE II.

FIG. 1.

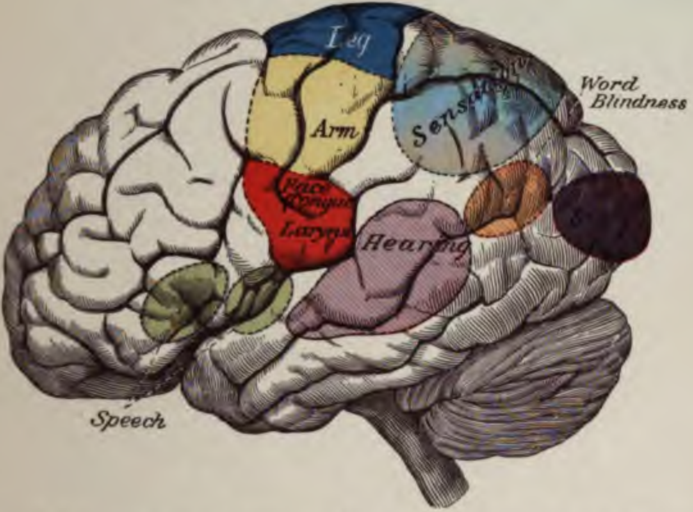


Chart of Localization of Cortical Centres determined on External Surface of Cerebrum. (Gray.)

FIG. 2.

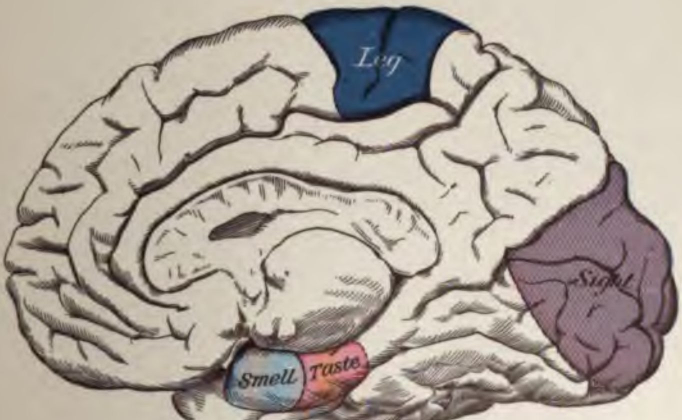


Chart of Localization of Cortical Centres determined on Medial Surface of Cerebrum. (Gray.)

Fig. 61.

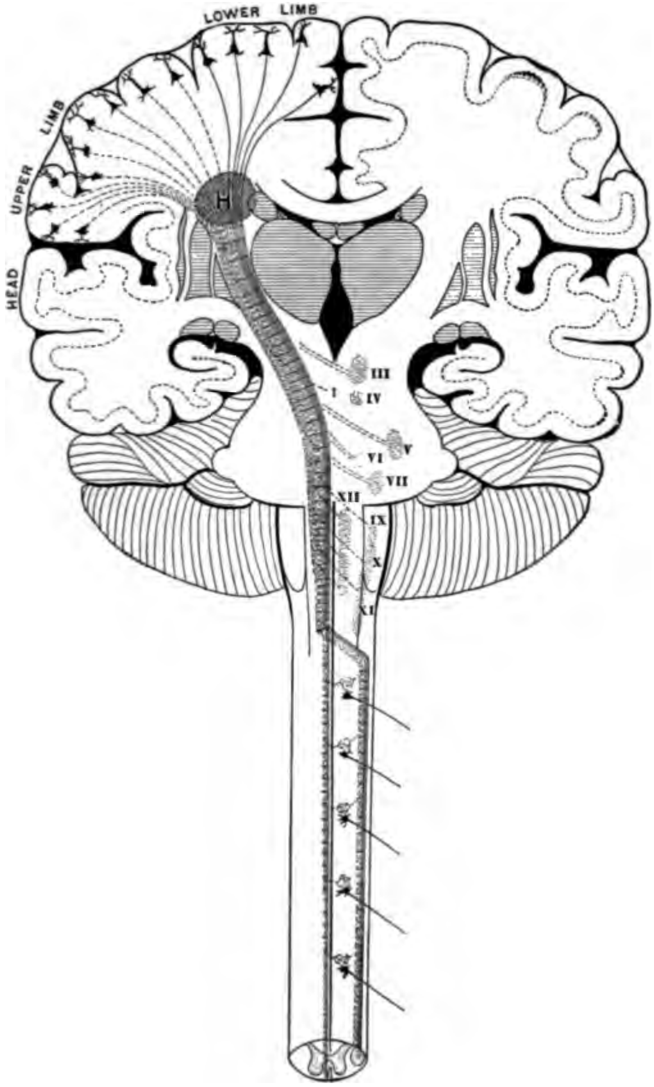
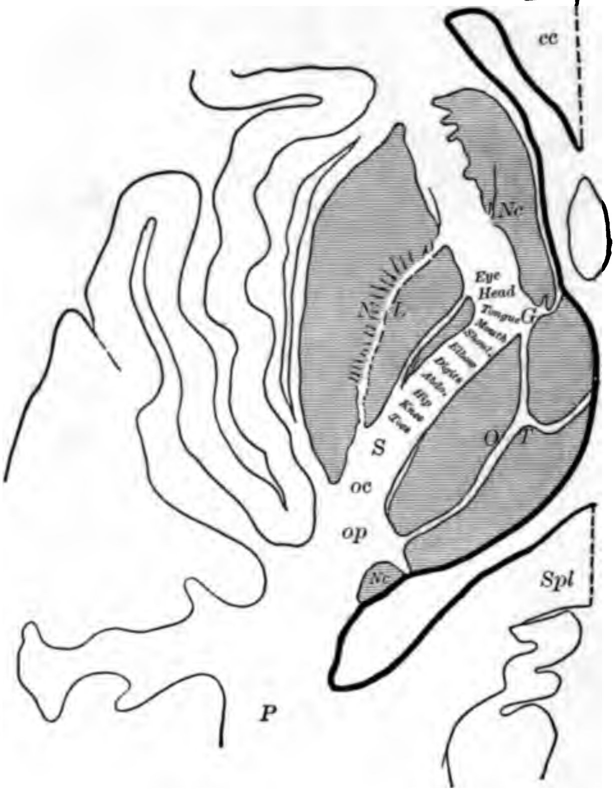


Diagram showing the fibres from the cortex forming the corona radiata, which after they are approximated pass into the internal capsule. It also shows the decussation of the pyramid of the left side, which passes to the right side of the spinal cord, and the direct or uncrossed tract (Turck's column). Finally it also shows the secondary degeneration which occurs after cerebral hemorrhage or softening, and which follows the course of the motor tracts into the spinal cord. H. Site of lesion. The continuous lines are fibres going to the legs, the dotted are those going to the arms and motor cranial nerves. (Modified from VAN GEHUCHTEN.)

FIG. 62.



Outline of horizontal section of brain, the fibres of the internal capsule being cut across. Natural size. The gray matter of the cortex and claustrum is left unshaded, but that of the corpus striatum and optic thalamus is shaded. *O T*, optic thalamus, showing the median, lateral, and anterior nuclei; *N L*, nucleus lenticularis, showing the putamen large, and the inner division of the globus pallidus very small; *Nc*, nucleus caudatus, the large head in front of and the diminishing tail near the thalamus; *G*, the knee of the internal capsule. From "*Eye*" to "*Digits*" marks the position of the pyramidal tract as a whole, and the several letters indicate broadly the relative positions of the several constituents of the tract, named according to the movements with which they are concerned: thus *Eye*, movements of the eyes; *Head*, of the head; *Tongue*, of the tongue, *Mouth*, of the mouth; *Shoul.*, of the shoulder; *Elbow*, of the elbow; *Digits*, of the hand; *Abdo.*, of the abdomen; *Hip*, of the hip; *Knee*, of the knee; *Toes*, of the foot; *S*, the temporo-occipital tract; *oc*, fibres to the occipital lobe; *op*, optic radiation. At this level the fibres of the frontal tract, in the fore limb of the capsule in front of the pyramidal tract, run almost horizontally, parallel with the plane of the section. *cc*, the rostrum of the corpus callosum; *Spl.*, the splenium of the same, both cut across horizontally. The thick dark line indicates the boundary of the cavities of the anterior and descending horns of the lateral ventricle and of the third ventricle, the two ventricles being laid open into one by the removal of the velum and choroid plexus, etc. The oval outline in the fore part of this cavity indicates the fornix. Lateral to the nucleus lenticularis are seen in outline the claustrum, the cortex of the island of Reil, and the operculum or convolution overlapping the island of Reil. *P.* is inserted to show which is the hind part of the section.

PLATE III.

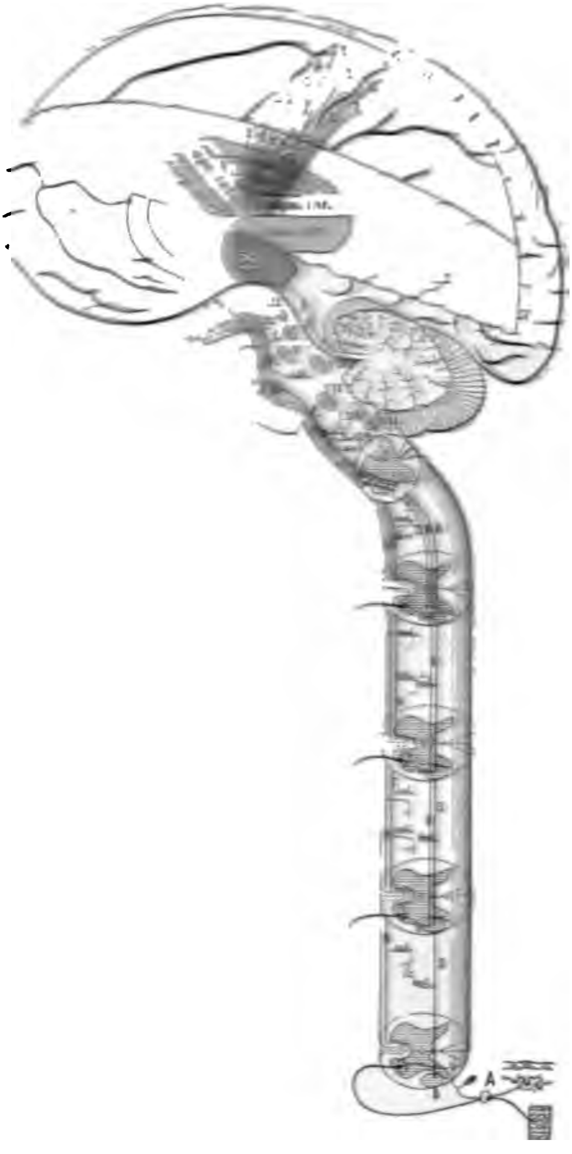
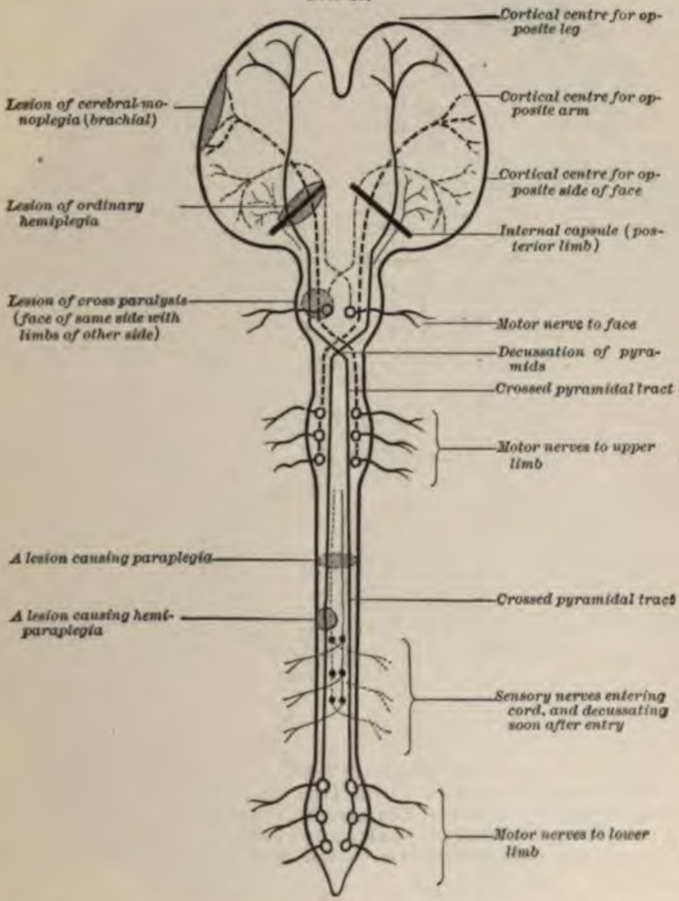


Diagram showing Course of Motor Fibres from the Cerebrum and Cord to the Periphery. (Flatau.)

function, and these fibres approximate one another more and more closely in the lower part of the brain until they form a bundle (the corona radiata). Thus we see in Plate III. how the fibres arising from the middle area of the cortex cerebri pass down through the lenticular nucleus into the knee or angle of what is called the internal capsule. This is a lateral view. In Fig. 61, which also shows the results of a lesion in the capsule, we get an antero-posterior view. These fibres are arranged in such a way that those arising from the lower part of the cortex, as in the face-centre, lie nearest the knee

FIG. 63.

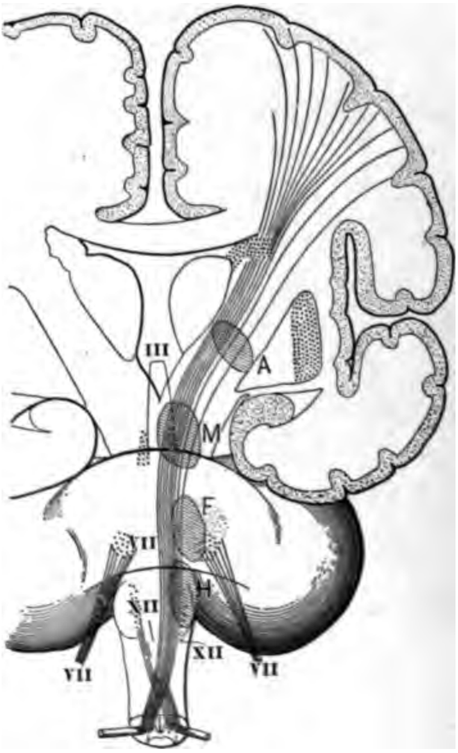


Cortical centre for opposite leg
Lesion of cerebral-monoplegia (brachial)
Cortical centre for opposite arm
Cortical centre for opposite side of face
Lesion of ordinary hemiplegia
Internal capsule (posterior limb)
Lesion of cross paralysis (face of same side with limbs of other side)
Motor nerve to face
Decussation of pyramids
Crossed pyramidal tract
Motor nerves to upper limb
A lesion causing paraplegia
Crossed pyramidal tract
A lesion causing hemiparaplegia
Sensory nerves entering cord, and decussating soon after entry
Motor nerves to lower limb

Diagram showing the general arrangement of the motor tract and the effect of lesions at various points. (ORMEROD.)

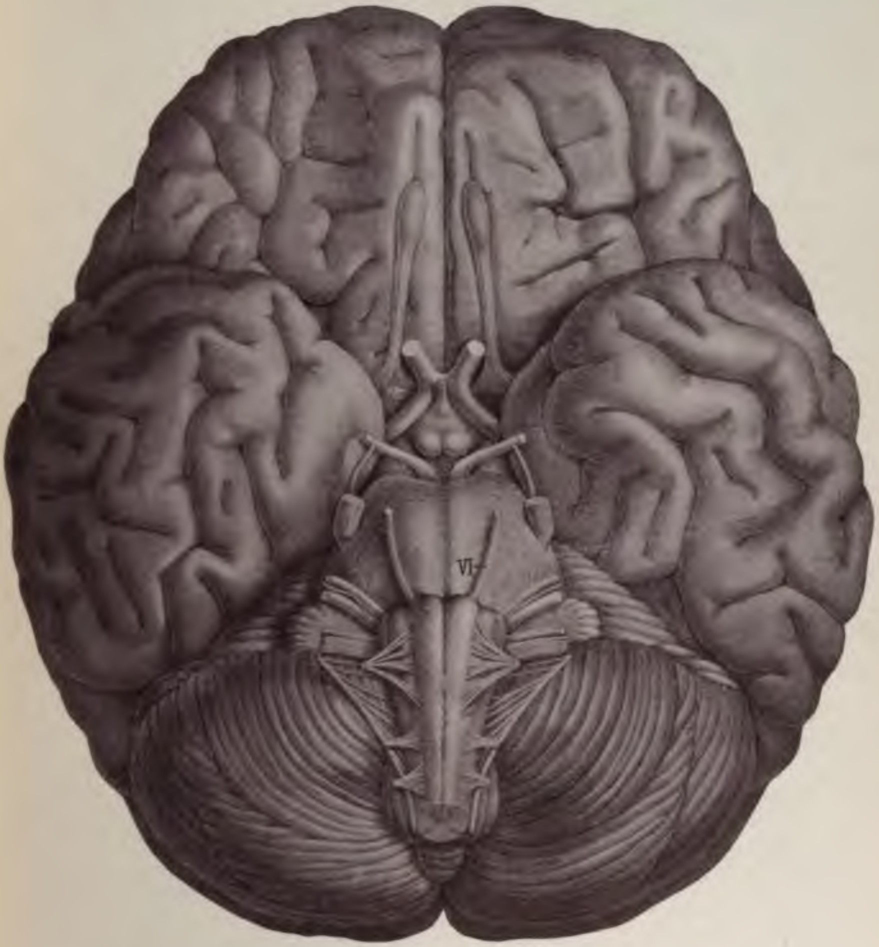
of the capsule, and those highest, furthest from this point. (Fig. 62.) After the motor fibres have passed through the internal capsule they pass into the crus cerebri of that side, which (the crus cerebri) connects the hemisphere of the same side with the cerebellum behind it, and the pons and medulla below it. The crura cerebri are two thick, cylindrical bundles of white matter which emerge from the anterior border of the pons (Plate IV.), diverge as they pass upward and outward to enter the under part of each hemisphere, as if stretching out to receive the motor fibres from the internal capsule. From the crura cerebri the motor fibres pass downward into the pons Varolii. Here the fibres which have hitherto travelled

FIG. 64.



Showing the mechanism of different hemiplegias. A lesion at A causes complete hemiplegia by destroying the motor tract. One at M causes paralysis of third cranial nerve (motor oculi) by destroying its nucleus or root on same side, and paralysis of arm and leg on opposite side. A lesion at F causes facial palsy on same side, hemiplegia on opposite side. In a lesion at H the hypoglossus would be affected on one side, with hemiplegia on the other. (Modified from EDINGER.)

PLATE IV.

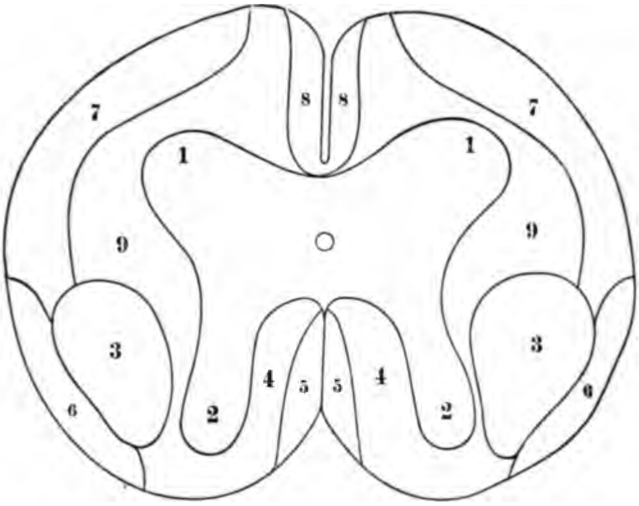


Base of Brain Showing Several of the Cranial Nerves, Notably the Sixth.

The crura cerebri are seen on either side of the posterior perforated space and under the third nerves. (Modified from Arnold.)

together divide into two parts, namely, those from face and tongue centre, which pass to the opposite side and become connected with the nuclei of the facial and hypoglossal nerves, which act as minor centres governing the face and tongue, and the fibres for the arm, leg, and trunk of the body, which continue on down to the medulla oblongata, where they form the so-called pyramids, and having done so most of the fibres cross to the opposite side of the spinal cord (the crossing of the pyramids), and so form the crossed or lateral pyramidal tracts. (Fig. 61.) A smaller number of fibres, however, pass directly down to the spinal cord from the medulla oblongata, and form what is called the direct or anterior pyramidal tract. Direct, because it does not cross; anterior, because it lies along the edge of the anterior fissure of the cord; pyramidal, because it comes down from the pyramid. This is sometimes called Türck's column. (Fig. 61.) It is by means of these two tracts in the spinal cord that motor impulses pass down to the nerve-trunks and muscles.

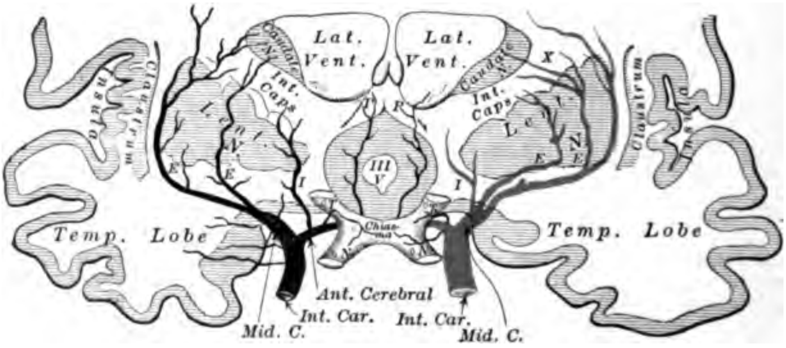
FIG. 65.



Showing tracts in spinal cord. 1. Anterior horns of gray matter, which contain the cells governing the nutrition of the muscles and give rise to the motor roots. 2. Posterior horns of gray matter, which receive the sensory roots. 3. Crossed lateral pyramidal or chief motor tracts from cortex of brain. 4. Columns of Burdach, or the outer sensory tracts, carrying impulses upward. 5. Columns of Goll, or inner sensory tracts, carrying impulses upward. Tactile sensibility. 6. Direct cerebellar tract, which carries impulses of a sensory character upward. Tract of muscle-sense. 7. Antero-lateral tracts, which consist of fibres conducting the gray matter of the cord into that of the medulla. They contain anterior nerve-roots and are the channels for reflex effects. There are also tracts in this root for pain and temperature sense. 8. Column of Türck, or direct anterior pyramidal tract, which carries impulses of motion downward. 9. Lateral mixed tracts same as 7.

We can understand, therefore, that if a small lesion occurs at the peripheral endings of the corona radiata—that is, on the cerebral cortex—it will only produce a limited paralysis. Thus, as seen in Fig. 61, a clot at the arm-centre would only involve the arm-fibres. If, however, the lesion be lower down where the fibres of the corona radiata are getting closer and closer, as, for example, in the internal capsule, then even a small lesion will produce widespread paralysis, since it will involve a large number of fibres running ultimately to widely separated areas in the body, and, if large enough, produce hemiplegia. (Figs. 61 and 64, lesion A.) If the lesion be situated in the pons on one side, it will cause facial paralysis on that side and hemiplegia on the opposite side of the body, because, as shown in the diagram (Figs. 63 and 64, M), it will, under these circumstances, destroy the facial fibres after they have crossed, and the remaining motor fibres before they cross. The various tracts, motor and sensory, in the spinal cord are shown in Fig. 65.

FIG. 66.



Showing distribution of bloodvessels to internal capsule. The artery marked *x* is the so-called artery of cerebral hemorrhage, and it is readily seen how its rupture destroys the fibres in the internal capsule. (Modified from GORDINIER.)

Hemiplegia from hemorrhage is characterized by sudden onset in most cases, by more or less mental disturbance and disorders of motion, sensation, and of the special senses according to the site of the leaking vessel. The skin-reflexes are apt to be markedly decreased and the deep reflexes increased, but the bladder and rectum are not usually paralyzed, although in the first shock of the accident there may be vesical and rectal incontinence. The mental disturbance usually amounts to a rapidly oncoming unconsciousness in hemorrhagic hemiplegia.

The question of the location of the lesion is very important. In the great majority of cases it is situated above the point at which the decussation of the motor fibres takes place in the medulla, and is, therefore, on the opposite side of the body from that on which the hemiplegia exists. If, however, the lesion be below the decussation, the paralysis and lesion are on the same side, as just described. The most common site for the lesion in hemiplegia is in the knee or posterior limb of the internal capsule, owing to the fact that the middle cerebral artery in one of its lenticulo-striate branches perforates the internal capsule, and ends in the caudate nucleus, and this artery is so commonly ruptured that Charcot has called it the "artery of cerebral hemorrhage." (Fig. 66.) If the hemorrhage does not involve the posterior third of the internal capsule, there are no sensory symptoms associated with the motor loss, but the paralysis will be practically universal on that side, involving the leg and arm, and the lower part of the face, so that the mouth is drawn toward the healthy side. (Explained by Fig. 62.) The symptoms associated with hemiplegia due to this cause often become very severe, because the hemorrhage is so profuse that the lateral ventricles become filled with blood, and from them the blood passes to the third and from there to the fourth ventricle, where, by pressure on the vital centres, it speedily produces death. In such cases deep unconsciousness, stertorous breathing, a slow, full pulse, and a flushed skin, becoming somewhat cyanotic, may be present. Recovery never occurs, for the secondary inflammation, or softening, following the outflow of blood produces fatal results, even if the patient survives for some days.

In cases in which the hemorrhage is very limited consciousness may be lost for only a brief period, and at most there may be only mental confusion. Often in mild cases there is a slight return of power in the affected side within a few days, and the temperature of the affected part, which has been raised, approaches the normal. Finally, after six to eight weeks the dominant symptoms consist in partial loss of power of the arm and leg, and the facial paralysis has perhaps entirely disappeared, although the tongue when protruded may tend to go over to one side. If the case does not pass to such favorable results, instead of recovery of power at this time there are developed contractures and secondary rigidity from degenerative processes extending to the pyramidal tracts. (See Fig. 61.) Hitzig has shown that these conditions are apt to be least marked in the morning. Wasting of the paralyzed muscles only ensues from the disuse, and not from true trophic change.

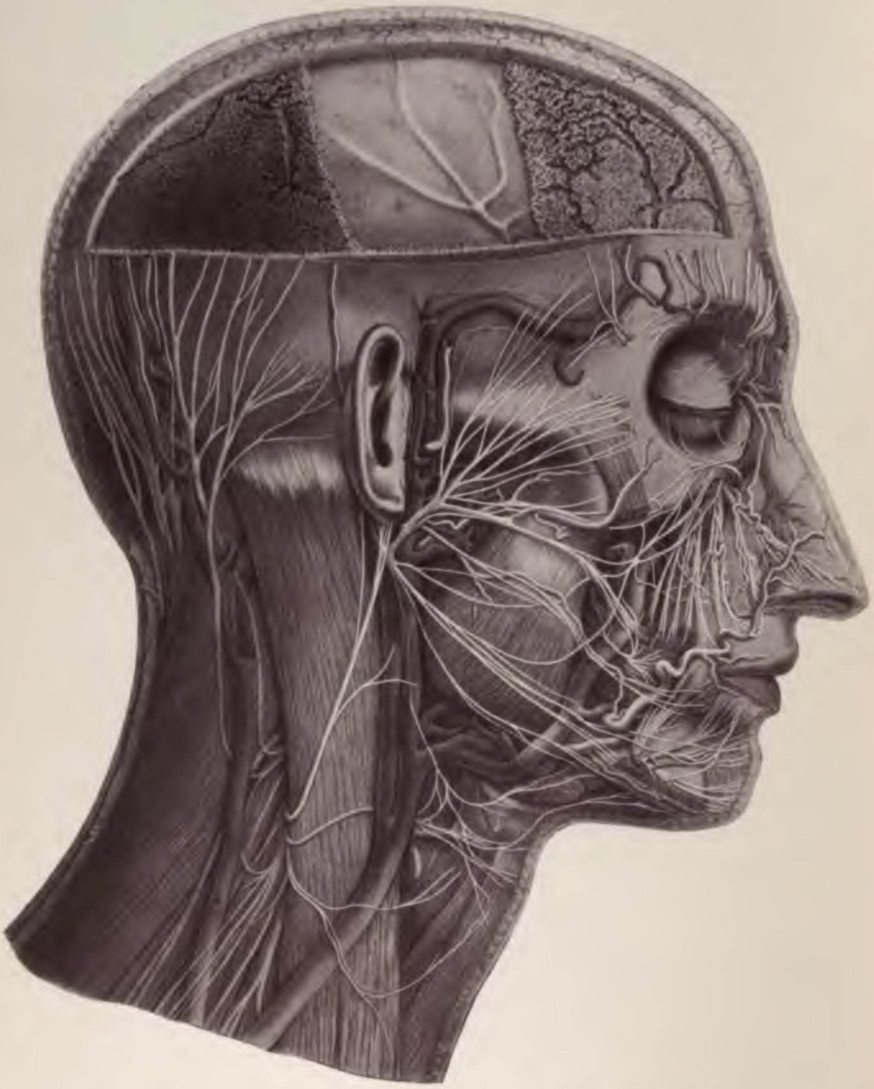
When the case is not of the very severe type which causes death in a few hours, and yet the lesions are such that recovery is not going to take place, the patient at the third or fourth day becomes unconscious a second time, his temperature rises, he mutters, and grows restless, finally becomes comatose, then develops respiratory failure, or a hypostatic congestion of the lungs, and dies.

When a patient is seized with headache, dizziness, vertigo, and vomiting, and rapidly oncoming hemiplegia and hemianæsthesia, attended at first with no loss of consciousness, but in a day by unconsciousness and coma, he is suffering from what has been called "ingravescent apoplexy." The hemorrhage, under these circumstances, begins in the knee of the internal capsule, proceeds backward till it involves the sensory fibres in the internal capsule, and, finally, breaks into the lateral ventricle, soon after which death ensues.

When a hemiplegia is followed by rigidity very early, with sensory involvement and convulsions, the lesion is probably cortical, or, more correctly, is secondarily cortical to a deeper hemorrhage, and spreads over the centres for the face, arm, and leg. Most commonly, however, cortical hemorrhages are due to injuries, or they may arise from unprovoked vascular rupture. In any case, they are usually ushered in by convulsions. (See next page.)

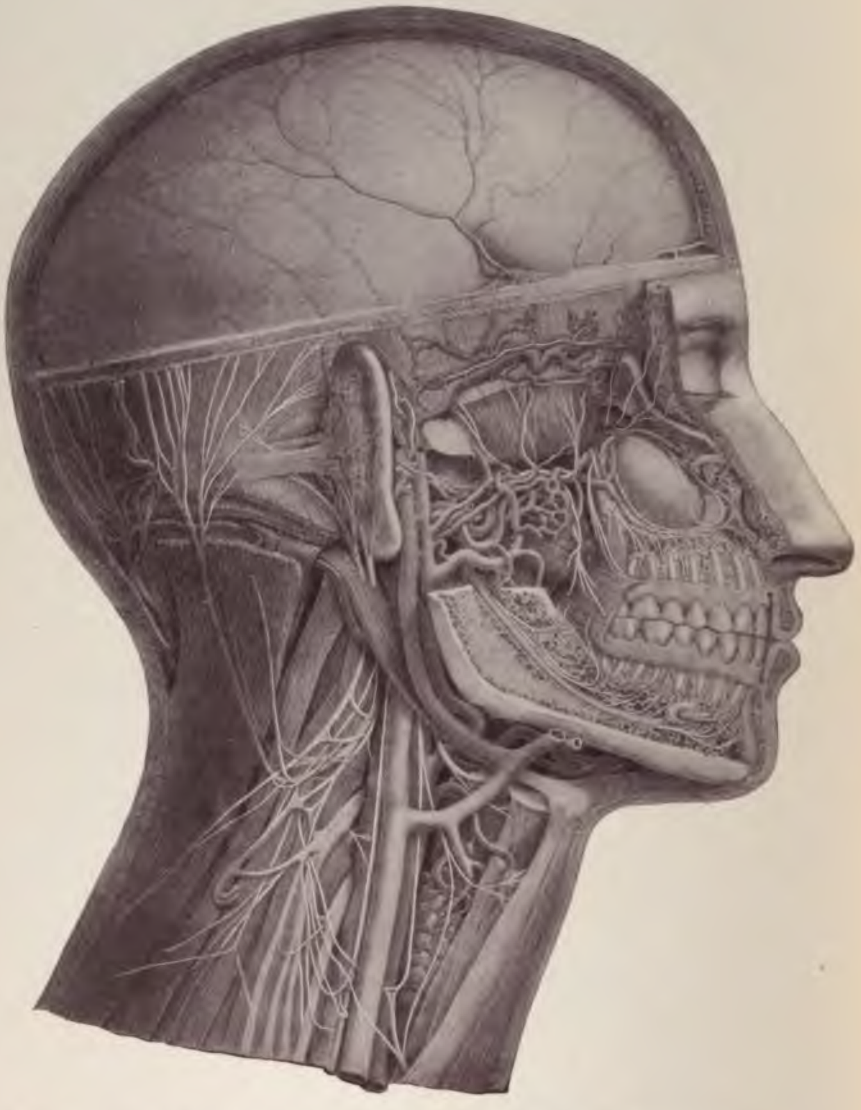
Where, on the other hand, there is paralysis of the arm, trunk, and leg on one side, with facial paralysis and anæsthesia on the opposite side of a well-marked type, associated with early rigidity of the paralyzed side, conjugate deviation of the eyeballs, very marked rise in bodily temperature, a contracted pupil, and convulsions, with difficulty in swallowing and in speech, the lesion is to be found in the pons Varolii on the side opposite the paralysis. This is due to the fact that the injury is below the decussation of the facial nerve. (Figs. 63 and 64.) If both sides of the face are paralyzed, with hemiplegia elsewhere, the lesion is in the pons where the facial fibres cross. Pons paralysis is nearly always associated with giddiness, vomiting, conjugate spasm with nystagmus, albuminuria, glycosuria, and marked disturbances in the respiration and heart. Pontile hemorrhages are, however, very rare, and usually are rapidly fatal.

PLATE V.



Showing the area of the middle meningeal artery, in the inner table of the skull, injury to which is sometimes the cause of hemiplegia. (Arnold's Atlas.)

PLATE VI.



The same Artery With the Inner Table of the Skull Removed.
(Arnold's Atlas.)

Finally, if there is hemiplegia involving the lower part of the face, the arm, and the leg, and in addition paralysis of the upper part of the face, and ptosis from paralysis of the facial and oculomotor nerves on the opposite side, and in association impaired sensibility and vasomotor changes in the limbs, the lesion is probably in the crus cerebri on the side of the upper facial paralysis—that is, on the same side as the ptosis; but this is only true if the two paralyses have been simultaneous in occurrence, as it is possible for one lesion in one place to produce paralysis of the face and another elsewhere to produce the hemiplegia (see Ptosis in chapters on Face and on the Eye).

If in the development of symptoms of cerebral hemorrhage there be little hemiplegia and temporary unconsciousness, followed in some hours by a sudden aggravation of the symptoms, it may be that in the beginning of the attack the lesion has been in the frontal lobes, but has gradually extended backward until it has ruptured into the lateral ventricle. So, too, a hemorrhage into the occipital lobe or the posterior part of the parietal lobe is rarely marked by much hemiplegia, and, if present, the leg is more paralyzed than the arm. The characteristic symptom, however, is well-marked hemianæsthesia (see chapter on Skin), and hemianopsia (see chapter on Eye). Generally, however, such changes result from a thrombosis.

Hemiplegia developing after an injury to the head is usually due to rupture of the middle meningeal artery or one of its branches (Plates V. and VI.). Owing to the fracture of the skull, the vessel is ruptured. A pathognomonic sign of this accident is the fact that there is a period of consciousness between the time of injury and the development of unconsciousness. The patient may be momentarily stunned, then recover consciousness, and then relapse into a second period of unconsciousness which rapidly deepens into profound coma. It is important to remember that while the paralysis is on the opposite side of the body from the hemorrhage, it may be on the opposite side from the injury, since the fracture of the skull may be a fracture by *contre-coup*. The paralysis is usually progressive from part to part, and convulsions may occur as a result of cortical irritation produced by the hemorrhage. A wide dilatation of the pupil on the side of the hemorrhage ("Hutchinson's pupil") may be present. Such a hemorrhage is said to be supradural. Sometimes the hemorrhage arises from the ascending frontal or ascending parietal artery, and is then subdural.

The following table, from Mill's *Nervous Diseases*, gives the differentiation of ventricular and meningeal bleeding:

Ventricular Hemorrhage.	*Meningeal Hemorrhage.*
No antecedents to explain the lesion.	Antecedents: for the newborn a prolonged or hard labor; for the adult a traumatism.
Premonitory symptoms not rare.	Premonitory symptoms generally absent.
Gradually deepening unconsciousness.	When traumatic, unconsciousness followed by partial restoration of consciousness and then again by unconsciousness.
Cephalalgia rare.	Cephalalgia frequent.
Paralysis when present is usually hemiplegic.	Paralysis ordinarily generalized; hemiplegia exceptional.
Varying pupillary conditions.	Unilateral dilatation of pupil common.
Deviation of the mouth and tongue very common.	Deviation of the mouth and tongue rare.
Contractures very common.	Contractures, although frequent, less common.
Convulsions may be present, but not common.	Convulsions the rule.
Vomiting not frequent.	Vomiting frequent.
No symptoms of secondary meningitis with fever.	Symptoms of secondary meningitis with high fever toward the third or fourth day.
Death rapid.	Life, as a rule, prolonged several days.

When there is developed in cases of hemiplegia, aphasia or disordered speech, there is probably a lesion in the neighborhood of the third frontal convolution, or the island of Reil (see chapter on Speech).

Hemiplegia may be due to cerebellar hemorrhage, in which case there are loss of consciousness deepening into profound coma, contracted pupils, vomiting in many of the cases, and finally death when hemorrhage breaks into the lateral ventricle. The diagnosis of cerebellar hemorrhage is very difficult.

Of the irregular forms of hemiplegia there are several. Sometimes the leg is from the beginning more affected than the arm, and remains paralyzed long after the face and arm have recovered. The leg may become rigid and distorted by contractures, and there will often be found present marked anæsthesia of the skin of the paralyzed leg and arm, with hemianopsia and aphasia. Such symptoms indicate a lesion of comparatively small size involving the leg-fibres and some of the sensory fibres in the internal capsule, and result from rupture of the lenticulo-optic artery. When the arm suffers most the symptoms just described as in the leg are seen in it, and motor aphasia, if the lesion is on the right side, is often very marked, as is also facial paralysis. This is supposed to be due to the anterior frontal artery, a branch of the inferior anterior cerebral artery, becoming diseased.

When post-hemiplegic chorea attacks the paralyzed limbs there is often a focal lesion in the posterior extremity of the internal capsule. The symptoms which have just been detailed may also arise, as already said, from embolism or thrombosis of the cerebral vessels as well as from hemorrhage from them. How are we to separate the hemiplegias of hemorrhage and occlusion? In many cases this is impossible, but there are some differential points which may aid us. In the first place, thrombosis is a condition of advanced age, while hemorrhage may occur at any time from thirty years of age on. The presence of hemiplegia in a young man, therefore, is probably not due to a thrombosis. Again, hemorrhage occurs often after exertion or the drinking of stimulants, and occurs rarely in sleep, whereas thrombosis not rarely comes on under these circumstances, and often develops during the night, so that the patient awakes paralyzed, but a patient may have both thrombosis and apoplexy. In hemorrhage, consciousness is generally lost, whereas in thrombosis it is often only dimmed. Vomiting and contracted pupils from pressure on the lower centres indicate hemorrhage, while their absence may point to thrombosis. Finally, the general systemic shock and febrile movement are apt to be greater in hemorrhage than in thrombosis. The history of syphilitic infection, producing an endarteritis, also points to thrombosis, although hemorrhage may arise from this cause also.

The diagnosis of embolism producing hemiplegia from the paralysis due to hemorrhage is always more or less difficult, but the presence of chronic or ulcerative endocarditis or their results, or other cause for the formation of emboli, aids the diagnosis. Where the cause is embolism the onset is sudden, whereas in thrombosis it is sometimes more gradual. The paralysis from embolism is more commonly on the right side of the body, owing to the fact that it is more easy for an embolus to pass into the left middle cerebral artery than into the right.

Spastic hemiplegia may be due to cerebral tumor, and is often associated with convulsions, particularly if the growth be cortical. Very often the paralysis of cerebral tumor will be found, from the history, to have come on gradually. Thus, the history may be that at first the side of the face has been paralyzed, then the arm and then the leg, and that the complete loss of power has not been sudden but gradual in the part affected, or that a convulsion has left that side, which was previously only impaired in strength, totally paralyzed.

Hemiplegia also comes on as a result of cerebral syphilis, and, aside from a history of specific infection and response to specific medication, presents few characteristic signs. The presence of intense headache, convulsions of an epileptiform type, and the fact that the paralysis occurs in some cases in early adult life, point to its origin.

Another cause of hemiplegia is diffuse cerebral sclerosis of one hemisphere (not multiple sclerosis), in which the most constant symptoms are, in addition to the paralysis, evidences of motor irritation, such as epileptoid convulsions of a bilateral or unilateral character, rhythmical or arhythmical twitchings of the muscles like chorea, and dementia.

Hemiplegia arising from acute infantile cerebral paralysis has many of the distinctive features already described when discussing the paraplegia due to this diseased state. The age of the patient, the occurrence of epileptiform convulsions and of athetosis in the affected parts, and the patient's history are the important points to be recalled in making a diagnosis. The lesion is always due to a cerebral hemorrhage or to embolic softening.

When hemiplegia occurs in locomotor ataxia, it depends not upon the disease, but upon a complicating hemorrhage, embolism, or thrombosis.

A slowly developed hemiplegia sometimes results from disseminated sclerosis, the pathological process involving the side of the pons and spinal cord, but the intention-tremor, the peculiar speech, the nystagmus, and the very excessive reflexes aid us in the diagnosis of this cause of the loss of power. (See chapter on Hands and Arms, part on Tremor.)

A form of hemiplegia which is often very misleading is that occurring in general paralysis of the insane or paretic dementia. In this disease the patient often has attacks of vertigo, unconsciousness, and more or less marked hemiplegia or monoplegia, sometimes with aphasia if the right side is paralyzed. This form is also liable to be wrongly diagnosed by reason of the epileptiform convulsions, which frequently occur, and which in connection with the paralysis give the impression in the first attack that there is a hemorrhage of the cerebral cortex. The altered disposition of the patient, the loss of memory and intelligence, the peculiar stumbling speech, and the curious changes in the handwriting are some of the symptoms which complete the diagnostic picture.

In this condition we should not forget the possibility of hemiplegia or monoplegia occurring with suddenness in the course of renal disease with uræmia. The paralysis may be permanent or only transient, but the urine will be found to be albuminous, and the other signs of renal disorder may be manifest.

Hemiplegia sometimes comes on in purulent meningitis. The history of a head-injury or of a pyæmic or infective process, the cerebral symptoms, the stiffness of the back of the neck, the impairment of the normal movements of the eyeball, and the optic neuritis, associated with the convulsions, make the diagnosis possible.

A very rare form of paralysis, in which the arm on one side and the leg on the other side are involved, is due to a bulbar lesion just where the decussation of the pyramids takes place. This is one of the forms of crossed paralysis, and is due to cutting off of one set of fibres before they cross, and the others after they have crossed. (See early part of this chapter and chapters on Hands and Arms, Feet and Legs, and Face and Head for further information as to crossed paralysis.)

CHAPTER V.

THE TONGUE, MOUTH, PHARYNX, AND ŒSOPHAGUS.

The general appearance of the tongue—Its coating—Its appearance in poisoning—Fissures and ulcers of the tongue—Eruptions on the tongue—Atrophy and hypertrophy of the tongue—Paralysis—Tremor and spasm of the tongue—Tonsillitis—Diphtheria—Pharyngitis—Disease of the œsophagus.

THE appearance of the tongue is recognized as indicative of the general condition of the patient, and is a valuable diagnostic aid in many diseases other than those associated with disorder of the gastro-intestinal mucous membrane. In examining this organ the physician should take note of the condition of its surface, its shape as it lies in the mouth or is protruded, and the character of its movements. He should also see that it is well protruded, and examine the back of it more than the tip, as the latter is the part giving the least information.

Before discussing the precise appearance of the tongue in the various disorders in which it becomes altered in appearance it is well to remember that its surface is covered by mucous membrane, which differs in various parts. The epithelium is scaly and rests upon the corium or mucosa. The mucosa also supports many papillæ, which are thickly distributed over the anterior two-thirds of the tongue on its upper surface. These papillæ give the peculiar roughness which is so characteristic of this surface, and occur in three forms, namely, the circumvallate or large papillæ, the fungiform or mediate, and the filiform. The circumvallate are only eight or twelve in number, and are arranged at the back of the tongue in the shape of the letter V, with the point toward the root of the organ. The fungiform papillæ are scattered freely over the tongue, mostly at the sides and tip, and appear as deep-red eminences, the bases of which are smaller than their free extremities. Their epithelial covering is very thin. The filiform papillæ, which cover the anterior surface of the tongue, are very minute, and arranged in lines corresponding in direction with the two rows of the circumvallate papillæ. From their apices project many fine, filiform processes which are of a whitish tint owing to the density of the epithelium of which they are composed. There are, in addition, many simple papillæ which cover the surface between the peculiar ones already described. The fungiform papillæ are those seen most commonly in cases of disease, for they become large and prominent, and because of their red color show through the coating as red dots.

The appearance of the surface of the tongue varies greatly even in health according to the condition of its mucous membrane and the epithelium covering it. The most common alterations in its appearance are due to mere superficial coatings or fur, which consist of dead epithelial cells, micro-organisms of many kinds, and abnormally shaped living epithelium. Small particles of food may also be present. Butlin believes that the coating is chiefly due to micro-organisms. The question as to how characteristic of a particular disease any one coating or fur may be has been warmly discussed. Some have gone so far as to assert that the coating of the tongue is not indicative of any state in particular, while others, of whom the author is one, are convinced that, while an absolute diagnosis of disease in other organs cannot be based upon the appearance of the tongue, great aid can be gained by its study.

There are, however, very few conditions of the coating of the tongue which are pathognomonic of any one disease, since the coating is produced by the local conditions of the mouth rather than by the disease itself.

Taking up for consideration the various forms of coating, we find that the area at the base between the circumvallate papillæ is always somewhat coated even in the best of health, and that in disease the heaviest coating is generally found in this region, while the tips and sides, even in those diseases in which the coating is heaviest, are generally fairly clear. This is in part due to the character of the epithelium in different parts, and to the fact that the tip and sides are generally scraped clean by the movements of the tongue. Further, it should be remembered that the development of coating, aside from digestive derangements, depends chiefly on three factors: first, immobility of the tongue, so that it is not kept clean by rubbing; second, mouth-breathing, whereby the surface becomes dry and less easily cleansed; and, third, fever, which not only dries the surface of the tongue by mouth-breathing, but interferes with salivary secretion. Additional local causes are a decayed or ragged tooth or follicular tonsillitis, which infects the lingual epithelium, lack of cleanliness, and habits, such as smoking. In the last class of patients, the smokers, a heavily coated tongue in the morning is very common.

The tongue of the typhoid state, and of typhoid fever in particular, is quite characteristic, because the prolonged illness, the great exhaustion, and the general apathy of the patient all conspire to produce a peculiar coating on this organ. Early in the disease the surface of the tongue may be more or less foul, resembling the coating associated with biliousness in that the back part is coated evenly and with a paste, but very soon a characteristic sign appears, namely, that the tip of the tongue and its edges become red, and the coating becomes most marked on each side of the median fissure, which increases in depth from before backward. The tongue also becomes narrow instead of broad and flabby, as it is in biliousness, and is drier. If the attack be mild, this condition may remain till convalescence is established; but if the disease runs a severe course, the coating becomes very heavy, more dry, rough and brown from exposure to air and medicine. The furred appearance becomes almost shaggy at the back portion, and the drying proceeds until the underlying epithelial layer is cracked and fissured, so that tiny exudations of blood add to the lingual discoloration. The reddened edges become dusky in hue, and may be cracked and fissured also. (Fig. 1, Plate VII.) The tongue is very slowly protruded on request, partly from mental apathy, partly from feebleness and because its surface is so stiffened that to move it is difficult. It is equally slowly withdrawn for similar reasons, and while protruded is often markedly tremulous. Toward the close of the attack the tongue cleans off through exfoliation of the dead epithelial accumulation, and this is a favorable or unfavorable sign according to whether the remaining surface is red and moist or dusky and dry. Sometimes these characteristic coatings do not appear, the tongue being brown and rough all through the disease.

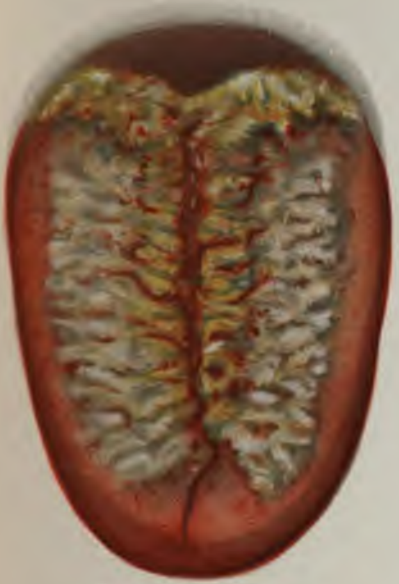
A small, triangular patch devoid of coating is often seen at the tip of the tongue in relapsing fever.

In uræmia the tongue is often dry, brown, cracked, and furred. The patient, if conscious, complains of a foul taste and the breath may smell like stale urine.

In biliousness the tongue is coated almost uniformly by a whitish-yellow, pasty coat, extending from back to tip and side to side. The tongue is broad and flabby, and sometimes indented by the teeth, while the breath is foul and heavy. (Fig. 2, Plate VII.) A simi-

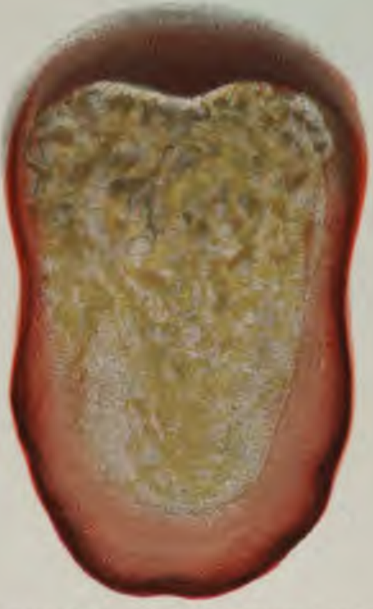
PLATE VII.

FIG. 1.



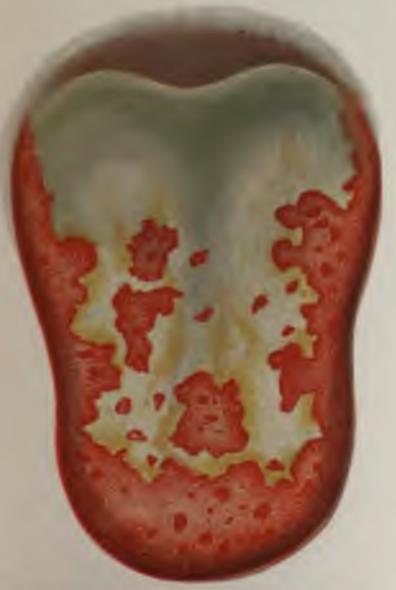
Typhoid Tongue.

FIG. 2.



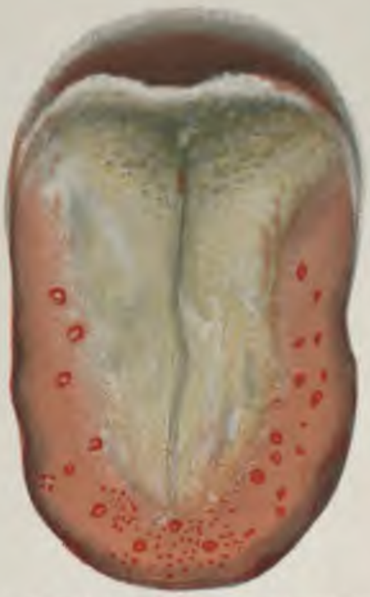
Bilious Tongue, with Yellow Coating.

FIG. 3.



Tongue of Mucous Disease or Chronic Catarrh of Stomach.

FIG. 4.



Tongue of Chronic Gastric Catarrh with Anemia.

lar tongue is seen in severe tonsillitis, except that it seems even more foul and less yellow in tint. Similarly in jaundice of the acute catarrhal type we have a coating still more yellow in some cases, because, as Fothergill asserts, the coat has been stained by the taurocholic acid eliminated by the salivary glands. The circumvallate papillæ are often prominent and stand above the coating, which is easily removed on scraping.

A broad, white, heavily coated, moist tongue is often seen in acute articular rheumatism, becoming dry if the fever is high and the attack prolonged.

The white tongue of a person who takes large amounts of milk is generally not smooth and pasty, but rather rough in appearance. If the tongue be suffering from an attack of thrush (saccharomyces albicans), the white coating will consist of irregular white masses of the growth, which, if in great number, often coalesce and make a fairly even surface. The soreness of the mouth, the local heat, the salivation, and the age of the person—generally a young child—render the diagnosis easy.

A grayish diphtheritic-looking coating of the tongue, occurring in adults, may be due to the growth of various forms of mycoses. Thus, a fine network of leptothrix in threads and tufts often spreads over the tongue, particularly in the region of the circumvallate papillæ. The growth may be quite dark in color, but it is separated from the exudate of diphtheria by microscopic study and the absence of systemic disturbance.

Sometimes on examining the tongue of a child we find that it is broad and flabby and covered by a gray coating, which is smooth and fairly moist. Scattered throughout this coating are patches in which the coating and epithelium have been shed, leaving red spots with sharply defined edges, which spots are said to be "worm-eaten" in their appearance—that is, to have the irregular outline of the marks on a worm-eaten leaf. In these areas are to be seen enlarged and reddened fungiform papillæ. Such a tongue is typical of what has been called, by Eustace Smith, "mucous disease," a condition in which there exists a more or less marked chronic catarrhal process in all the mucous membranes. (Fig. 3, Plate VII.) If, on the other hand, there is a comparatively light coating, dotted irregularly by bright-red spots, which are not raised above the surface, but are very numerous, and the patient is a child, the diagnosis may be made of acute or subacute gastric catarrh. (Fig. 4, Plate VII.)

The so-called strawberry tongue is one in which the organ is entirely denuded of coating and superficial epithelium, while the fungiform papillæ are swollen or enlarged and stand out prominently. This appearance of the tongue is seen commonly in scarlet fever, but is not, as has been thought, pathognomonic of that disease. The fungiform papillæ in the strawberry tongue of scarlet fever are, however, particularly prominent and erect.

When the tongue is excessively furred or rough in appearance, the coating is due to abnormally long and projecting papillæ covered by an excess of living and dead epithelial cells; it may denote grave disease of the viscera, but in rare instances possesses no diagnostic importance, unless coupled with other symptoms. This tongue is sometimes seen in scrofulous children in whom strumous manifestations are marked.

Should the tongue be denuded not only of coating, but, in addition, of its normal epithelium, so that it appears dry, hard, and harsh to the touch, it denotes, as a rule, grave and advanced disease of an exhausting nature, such as renal, hepatic, or gastric disorder about to cause the death of the patient. Sometimes this condition is seen in advanced phthisis, diabetes, or gastric carcinoma, and is of evil omen. When the tongue is bereft of epithelium, beefy and red looking, elongated and narrowed, and shows a peculiar roundness when protruded, severe disease of the abdominal organs, such as dysentery, or hepatic abscess, or carcinoma, will often be found, or, in some cases, this condition develops to add to the discomfort of cases of advanced pulmonary tuberculosis or acute peritonitis. This tongue is sometimes called the "parrot tongue."

In this connection the point should be noted that dryness of the tongue in the presence of grave disease is always an evil omen, and returning moisture of the tongue a favorable one.

Unilateral coating of the tongue may be due to a decayed or ragged tooth, or to hemiplegia, which prevents that side of the tongue from being cleaned through movement. Hillow and Fairlie Clark both assert that morbid conditions of the second division of the trifacial nerve cause unilateral coating, and that abnormalities of the third division do not produce these changes as we would expect.

The coating of the tongue is often so stained by extraneous substances as to be entirely changed in appearance. If the coating be black, the color may be due to the ingestion of iron, of bismuth, charcoal, ink, or blackberries, mulberries, cherries, or red wine. In very rare cases it is black, not from the growth of a fungus, as has been thought, but from overgrowth of the epithelium with the deposit of a black pigment of unknown origin. Usually this brownish-black discoloration is confined to the middle of the tongue. The affected surface is often rough, due to the enlarged papillæ, and the edges of the spot are less black than the centre. In professional tea-tasters the tongue may be orange-tinted.

The coating may be stained brown from the chewing of tobacco, from licorice, nuts, prunes, or chocolate, and yellow from the ingestion of laudanum or rhubarb.

The color of the tongue itself, aside from discoloration of its epithelium, is an important diagnostic aid. It is exceedingly pale in all forms of anæmia, particularly those due to lack of hæmoglobin, such as chlorosis or acute anæmia from hemorrhage, and in pernicious anæmia, when well advanced, it has a remarkable pallor. It is livid and cyanotic in cases of pulmonary disease interfering with oxidation of the blood, or in cardiac disease with similar difficulty.

Purple spots, which may be almost black, may be present on the tongue in Addison's disease. Sometimes they are bluish-black, and always well defined and even with the surface. Very rarely the tongue is discolored by infarcts, blood-stains, and bruises. When the tongue has its edges dotted with yellowish patches of a slightly elevated character, the condition is xanthelasma, and the liver will often be found to be disordered.

In cases of poisoning by corrosive sublimate the tongue presents a most characteristic appearance, for it is white and shrivelled, and the papillæ at the base are unusually large.

When sulphuric acid has been swallowed the tongue has a parchment-like appearance, is at first white and then gray or brownish-gray, and finally is covered by a black slough, which as it separates leaves a swollen, excoriated patch. In nitric- and chromic-acid poisoning the tongue is shrivelled and lemon-yellow in color, as it is when hydrochloric acid has been swallowed. The tongue of carbolic-acid poisoning is very characteristic indeed, for the mucous membrane is shrivelled and puckered into folds. The spots where the acid has touched it are brownish if impure acid has been swallowed, or white if the pure acid has been taken. In the course of a few hours this spot becomes surrounded by a red zone, and finally becomes dark brown or black in the centre. After oxalic acid is taken the tongue may be covered by a thick white coat and looks as if it had been scalded. Caustic potash and soda soften the mucous membrane, so that it is pulpy and easily detached, and looks pearly, red or yellow in hue. When ammonia is swallowed the color is white, but superficial œdema may make it pearly in appearance, and acid nitrate of mercury renders it very red. Cantharidal poisoning produces large lingual blisters and sores.

Aside from the coating and color of the tongue, its surfaces should be examined to discover fissures, cracks, ulcers, sloughs, and swellings. The tongue is often seen to be superficially and irregularly fissured in old persons, particularly in those who have used large quantities of strong alcoholic drinks or strong tea, or who have chewed tobacco incessantly for many years. The fissures cross each other in every direction, although the central fissure which runs longitudinally is generally deepest and longest. If the furrows are very deep, they may indicate the early stages of what Wunderlich has called dissecting-glossitis, which in turn may be due to syphilis,[1] although, as a rule, the fissures of the tongue due to syphilis are deepest at the edges of the organ, and are due to pressure by and from irritation from the teeth or to ulceration, and subsequent cicatrization of small syphilitic nodules or gummata. The cervical glands are rarely involved in such cases. If only one ulcer is present it may be chancre, which will have the peculiar Hunterian hard base, and, in such a case, the cervical glands will probably be enlarged. An epithelioma may also have an indurated base with secondary glandular enlargement. Lingual ulcers may also be present as the mucous patches of syphilis, or be due to wounds from the teeth, a broken pipe-stem, or a fork. When these become chronic their separation from those due to syphilis and tuberculosis is practically impossible on superficial examination. Sometimes an ulcer of the tongue is due to epithelioma; but if this is the case, the patient will probably be past thirty years of age. As deep syphilitic ulcers heal sclerosis of the tongue may develop.

Multiple ulceration of the tongue may be due to tubercular disease, which is very rarely primary, but rather secondary to its presence elsewhere. The sores are often stellate in shape, and there is always swelling of the cervical lymphatics, whereas in multiple syphilitic ulceration of the tongue the glands generally escape. The diagnosis between tubercular ulcer and that due to epithelioma is more difficult, since in both diseases the cervical glands are involved. Both are more common in men than in women. The age of the patient, the presence of tubercular disease elsewhere, and the absence of induration point to tubercle. The tuberculous ulcer is not surrounded by much inflammation, is covered by grayish purulent mucus, and may contain bacilli of tubercle, and is often associated with tubercular nodules which have not broken down.

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[1] This is denied by Demarquay and doubted by Butlin.

Ulcers of the tongue may also be due very rarely to lupus. A very similar tongue is seen in a tropical disease with intestinal disorder called by Thin "psilosis." An herpetic eruption appears on the tongue, which leaves large areas devoid of epithelium, while sinuous furrows or fissures develop. These fissures then heal, the patches become pallid, and recovery takes place.

The various ulcerated surfaces so far described might be confused with ulcerative stomatitis, but their chronic character and insensitiveness as compared to acute ulcers of the tongue, associated with a specific history or manifestations of tuberculosis or syphilis elsewhere, render the diagnosis clear.

An ulcer on the frænum may be due to whooping-cough, in which disease the edge of the lower incisors may injure the tongue in the paroxysm of cough, or it may indicate the presence of a ragged tooth, which produces constant irritation, or, if the patient is advanced in years, represent the early stages of epithelioma, or that a broken pipe-stem has produced a wound.

Very rarely the tongue partakes of the ulceration of the tonsils and roof of the mouth which is seen in cases of Schönlein's disease, accompanied by purpuric eruptions on the skin and evidences of septicæmia.

Should the tongue be marked by bites from the teeth the patient may be an epileptic. Even if he denies that he is affected by the disease, the attacks may be unknown to him, because they are nocturnal. If the tongue is frequently bitten, the patient may be suffering from the early stages of glosso-labio-pharyngeal paralysis.[1]

The surface of the tongue may be attacked by various eruptions, such as measles, variola, eczema, herpes, erysipelas, pemphigus, zoster, or hydroa, and from the rupture of the vesicles or bullæ so formed ulcers may arise.

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[1] It may be pointed out in passing that if there be fits, and biting of the tongue never occurs, and the patient is a female, the attacks are probably hysterical.

If the sore is herpetic, de Mussy asserts that the eruption will be found in the distribution of the lingual branch of the chorda tympani along the under border at the side.

Sometimes the surface of the tongue is here and there devoid of epithelium, and in some of these patches excoriated. Pain may or may not be present. The condition is called chronic superficial glossitis by Hack, and is considered by some to be the same disease described by Kaposi as glossodynia exfoliativa. It is more common in men and lasts many years.

Urticaria of the tongue has been reported by Laveran and xeroderma pigmentosum by Keating.

The presence of a plaque on the anterior portion of the dorsum of the tongue to one side of the median line, which is raised, not ulcerated, but red and irritated-looking, may be due to excessive smoking, the smoke irritating the local epithelium. It is always very smooth, later covered by a yellowish-brown coat, and is sometimes called "smokers' patch." It may extend over the whole tongue and last for years.

When the tongue has on its dorsum and edges dull-white or slate-colored dots, patches, or lines, which are elevated, hard and horny to the touch, but not painful, the condition is known as leucokeratosis buccalis, or leucoma or ichthyosis, and this may arise from smoking or glass-blowing. It rarely begins in persons under twenty or in those over sixty years. It is often a strong predisposing agent toward cancer of the tongue. These spots are arranged on the tongue in longitudinal lines. Hyde asserts that they are due to excessive keratinization of the epithelium covered by an adherent and dense pellicle. The history is chronic, and ultimately by the stiffness of the spots the tongue may become cracked, and this in turn, perhaps, give rise to carcinoma. When the tongue is covered by smooth, dense plaques and disks or rings, the condition may be lichen planus, but the diagnosis of lichen planus from leucokeratosis buccalis is difficult, if not impossible. The plaques are most commonly seen in males between twenty and forty years. Closely allied to this is the rare condition of hardening of the tongue due to scleroderma, as described by Kaposi.

A very rare condition of the tongue is one in which its surface is marked by rings or areas on the dorsum, which gradually enlarge until they reach the edge or coalesce. In appearance they are red and smooth, deprived of filiform papillæ, but not of the fungiform variety. Often the border of the circle is more red than the centre, and the very edge is often yellowish. This condition is sometimes called wandering rash, geographical tongue, or annulus migrans. Little if anything is known of its cause, save that delicate children are most often affected by it.

Feeble, sickly children sometimes develop upon the tongue, as well as on the lips and cheeks, a condition in which a tenacious exudation is thrown out, the mucous membrane becoming fissured and sore. Gaston and Sebestre have called this stomatitis impetiginosa.

Œdema of the tongue, with the development upon it of vesicles, and, finally, sloughs, may occur, and is probably identical with the foot-and-mouth disease of domestic animals.

Bilateral atrophy of the tongue is due to disease affecting the hypoglossal nerves in some part of their course in or below the nuclei (see Paralysis of the Tongue). It occurs as a symptom of glosso-labio-pharyngeal paralysis, in which case the tongue is shrivelled and atrophied in patches, and in the later stages of the disease the organ has a crenated appearance. In other cases it is present in progressive muscular atrophy, and rarely in locomotor ataxia. It has also been seen in general paralysis of the insane. Unilateral atrophy may also occur from these causes, and Remak asserts that it sometimes arises from chronic lead-poisoning. Any disease involving the hypoglossal nerves may so result (see Paralysis of the Tongue).

In cases where the tongue is much enlarged the increase in size may be due to malignant growth, to macroglossia, which is a form of congenital lymphangioma, inflammatory hypertrophy, and syphilis, or acute inflammation from irritant poisons or foods. It may also be due to dermoid cysts, fibroma, lipoma, papilloma, angioma, myxoma, osteoma, and enchondroma. When it is due to acute glossitis the organ is seen to be several times its normal size, is protruded from the mouth, and marked by the pressure of the teeth. The organ is also clumsy and stiff, and heavily coated on the back portion. There is a profuse flow of saliva, and swallowing and speech are almost impossible. Glossitis may also be due to mercurialism, to septic infection, and may be either unilateral or bilateral.

The tongue may be greatly enlarged by actinomycosis. Great enlargement of the tongue may also arise in acromegaly and in myxœdema. In the latter disease the organ is broad, flat, and soft.

The movements of the tongue depend upon its innervation and its muscles, and afford valuable information in diagnosis. The rapidity of its protrusion in nervous and excitable persons when they are asked to show the tongue is noteworthy, and its constant rolling is often seen in persons who are feeble-minded. In all diseases associated with mental hebetude its protrusion on request is very slow, although the patient will often do this act when all other orders to move parts of the body fail to produce a response. In the various forms of coma due to apoplexy, diabetes, uræmia, and cerebral congestion, this condition obtains, and it is very characteristic of typhoid fever. Often the tongue which has been partially protruded is left so, even when the patient is told to draw it in. When the patient finds it difficult or impossible to remove food from between the teeth and cheek by means of his tongue, and complains that the power of speech is interfered with, because the tongue is clumsy in its movements, he may be suffering from the disease known as glosso-labio-pharyngeal paralysis or progressive bulbar paralysis. These lingual disorders are often the earliest signs of the disease. More rarely this disability of the tongue may arise from pseudobulbar paralysis, or what has been called glosso-labio-pharyngeal cerebral paralysis, a disease in which foci of softening occur in that portion of the cortico-muscular tract in which are the fibres which supply the muscles used in swallowing and speaking. This false type is separated from the true bulbar palsy by its sudden onset, an apoplectiform seizure, and other evidences of cortical disease. The tongue affords the most important points for differential diagnosis when a differential diagnosis is to be made under these circumstances, for in the false disease it does not waste or develop the reactions of degeneration, whereas in true bulbar paralysis these changes always speedily develop.

Paralysis of the Tongue. In apoplexy the tongue is protruded toward the paralyzed side, as it is also in the condition, already described, of hemiatrophy. The lesions of the hypoglossus which produce paralysis may be of cortical origin (unilateral), in which case the hemorrhage or other injury may be situated where the middle and inferior frontal convolutions form the anterior central convolution,[1] or in the supra-nuclear tract between the cortex and the medulla, or in the hypoglossal nucleus, or, again, in the infra-nuclear tract within the medulla. Insular sclerosis may very rapidly cause lingual paralysis. Paralysis of the tongue may also result from injury to the hypoglossal fibres outside the medulla through meningitis or syphilitic or other growths. In still other cases pressure upon the nerve in its foramen may cause unilateral paralysis, or wounds of the neck, caries of the first cervical vertebræ, or cervical tumors may so result. Often in such a case the spinal accessory nerve is also involved. Very rarely, indeed, the tongue may be paralyzed by a hypoglossal neuritis (Erb). In rare instances hemiatrophy of the tongue is associated with hemiatrophy of the face without hypoglossal injury (Gowers). Girard asserts that the sensory part of the trifacial contains trophic filaments for the tongue, and that the unilateral wasting may be due to disease of this nerve.

In paralysis of the facial nerve the tongue may be partially paralyzed through the fact that the lingualis muscle is supplied by means of the chorda tympani nerve. When a tongue which is paralyzed unilaterally is retained in the mouth, it is seen that its root on the paralyzed side is higher than the other, owing to the paralysis of the posterior fibres of the hypoglossus, but when it is protruded the tongue goes toward the paralyzed side because it is pushed out by the fibres of the genio-glossus muscle on the well side. Finally, let us remember that if the tongue is paralyzed on one side the lesion is in the cortex or the pons on the opposite side of the body, or in the nucleus in the medulla on the same side of the body, or in the nerve after it has left the medulla. If it is bilateral paralysis the lesion is probably nuclear, because the nuclei are so closely situated that even a small lesion involves both of them, or it may be due to symmetrical disease of both sides of the cortex, the so-called pseudo-bulbar paralysis already spoken of.

It should not be forgotten that paralysis of the tongue may occur as the result of diphtheria.

Hirt asserts that the reaction of degeneration may be found in the tongue whether the lesion be cortical or in the nucleus. If the lesion is only cerebral, this reaction will probably appear very late.

A tremor seen in the tongue may indicate a variety of nervous ailments or severe acute disease, as in typhoid and other severe infectious diseases, but the freedom from excessive coating and the absence of the ordinary signs of acute illness will separate the case of tongue tremor of acute disease from the tremor representing nervous ailments.

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[1] This is probably a fact, but not yet confirmed by autopsy, unless we consider Edinger's case of softening under this area, which affected the tongue only, as a typical one.

An important point to be regarded in noting lingual tremor is whether the tremor or fibrillary movement is constant, or whether it appears only when the tongue is moved to and fro or protruded. In typhoid fever the tremor occurs on movement, whereas in glosso-labio-pharyngeal paralysis when the mouth is open fibrillary movements of the organ are often marked, while the organ lies in the floor of the mouth powerless and beyond the control of the patient. Tremor of the tongue is also seen in a marked form in many cases of alcoholism, and associated with this tremor it will be noted that the protrusion of the organ is uncertain or in jerks.

Spasm of the tongue may be unilateral or bilateral, most commonly the latter. It is seen very commonly in cases of chorea, particularly of the post-hemiplegic type, and in hysteria. In the first disease the movements are characteristically choreic. In the latter the spasm may be tonic or clonic or alternately tetanic and irregular.

Often the spasm in hysteria is unilateral. Sometimes it is clonic in puerperal melancholia. Spasm of the tongue is a common symptom in association with the twitching of the lips of general paralysis of the insane. Jerky movements of the tongue may also occur in insular sclerosis, but this is not the cause of the peculiar speech of that affection.

Very rarely the condition of lingual spasm is due to irritation of the hypoglossus by some cause as yet unknown. The tongue is darted in or out or thrown from side to side and often injured by the teeth. The spasms, as a rule, are not constant, but come on in attacks which closely resemble epilepsy, in that they are preceded by an aura (Remak and Berger). A very rare affection termed aphthongia (Fleury) is characterized by spasm of the tongue on attempting to speak. Romberg has recorded a case of lingual spasm due to irritation of the fifth nerve from lingual neuralgia.

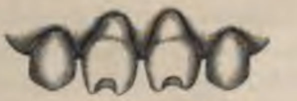
In that very rare condition called "Thomsen's disease," "characterized by tonic spasms in the muscles during voluntary movements," the tongue may be involved, but in this case the other voluntary muscles will share in the affection.

Having considered the diagnostic significance of changes in the appearance of the tongue in this chapter, and of the appearance of the lips in the chapter on the Face and Head, there is yet to be discussed the condition of the buccal mucous membrane, the tonsils, the soft palate, the teeth, the upper part of the pharynx, and the post-nasal spaces. As almost all the conditions found in the latter regions are of interest to the rhinologist rather than the general practitioner, only one or two affections of these parts will be included in this work.

We can sometimes gain some information from the teeth as to the state of the patient. Normally the two lower central incisors are cut about the sixth to the eighth month, then the four upper incisors from the eighth to the tenth month, and the lower lateral and all the front molars from the twelfth to the fourteenth month. The canines are cut from the eighteenth to the twentieth month, and the posterior molars at two to two and one-half years. The first permanent teeth usually begin to come in about the sixth year. In children who are sufferers from rickets the teeth decay very early and rapidly, and if they be sufferers from inherited syphilis, the teeth are often cut in the early months of life.

Caries of the teeth to an undue extent is also seen in many pregnant women and in cases of diabetes mellitus.

FIG. 67.



Hutchinson teeth.

If the permanent upper incisors are notched or peg-shaped with notches in the free edge, as if cut out with a small gouge, they are a fairly sure indication of syphilis of an hereditary character (Hutchinson teeth), and if in association with this deformity of the teeth we find middle-ear catarrh and keratitis, we have the "syphilitic triad," which is infallible as a sign of hereditary syphilis. These notches are not found in the so-called "milk-teeth."

The staining of teeth by tobacco or other materials held in the mouth may reveal certain habits of the patient, and a blue line on the gums where they join the teeth is an indication of the presence of chronic lead-poisoning. Loosening of the teeth, with bleeding, spongy gums should call to the physician's mind the possibility of scurvy or scorbutus, and the spongy gums are particularly indicative of this affection in bottle-fed babies. If loosening of the teeth occurs in adults, it may be due to mercurial salivation.

Grinding of the teeth in sleep in children usually indicates gastro-intestinal irritation from indigestion or worms, and it is sometimes seen in the advanced stages of respiratory diseases, as from pneumonia or diphtheria associated with dyspnœa. It takes place in adults in hysteria, maniacal attacks, and in epilepsy.

Difficulty in swallowing may arise from involvement of the pharyngeal muscles in diphtheritic paralysis, or from glosso-labio-pharyngeal paralysis (see chapter on Face, or retro-œsophageal abscess). Much more commonly it results from tonsillitis or pharyngitis. Not rarely it is due to a stricture of the œsophagus, and sometimes to a morbid growth in the walls of this tube, or to the pressure of such a growth situated in the surrounding tissues. If the difficulty in swallowing is due to diphtheritic paralysis the history will be that there had recently been an attack of diphtheria. If due to a lesion of the bulb there will be the symptoms described in the chapter on the Face, as referred to above. The presence of an inflammation of the pharynx or the tonsils is easily discovered by observation of the back part of the mouth, as is also retro-œsophageal abscess, which will generally be found associated with disease of the cervical vertebræ. If these states be excluded the diagnosis now lies between a stricture and a growth, and as the growth may be an aneurism the patient's chest should be carefully examined and the other signs of aneurism sought for, for should this be overlooked and an œsophageal sound passed, the aneurism, if present, may be ruptured. This examination may also disclose the existence of a mediastinal growth or enlargement of the retro-œsophageal glands. If these causes be eliminated the actual search for stricture may be begun. First the physician should listen over the cardiac orifice of the stomach while the patient takes a swallow of water. If the act of swallowing is properly performed this single swallow of water will be heard to descend to the cardiac orifice, and then pause there for six seconds before it falls into the stomach. If there is a stricture this fall will be delayed ; if there be atony of the cardia it will be hastened. An ordinary œsophageal bougie may be passed. If a point of resistance is discovered that part of the bougie-stem opposite the upper incisor teeth is marked, and then the instrument is withdrawn. In this manner we are enabled to tell the part of the œsophagus affected. Usually pain will be felt where the bougie is arrested; but care must be exercised that spasmodic contraction of the œsophagus is not mistaken for stricture. In the former state gentle and persistent pressure will overcome the obstruction. Usually the stricture exists at a point about six inches from the teeth; or where the left bronchus crosses the gullet, about eight or nine inches from the teeth; or at the cardiac orifice, which is seventeen inches from the teeth in the adult.

If a stricture exists it may be due to a cicatrix the result of an old burn, from the ingestion of alkalies or acids, or from an ulcer due to another cause. In other cases the lesion is due to syphilis.

If the obstruction be due to cancer the passage of a bougie may do great damage, and, therefore, if any intimation of the existence of such a growth is present, great gentleness must be used. It should also be remembered that the bougie may be arrested by its passage into a diverticulum, or, in other cases, the instrument, by coiling on itself, may give a wrong impression as to the site of the obstruction. If a diverticulum is present the food which is obtained from it is usually alkaline, as it has never entered the stomach, and milk derived from a diverticulum, in which it has tarried a short time after attempted swallowing, will not be coagulated.

In doubtful cases it is advisable to have the patient swallow a large dose of bismuth in mucilage of acacia and then examine the œsophagus with the Röntgen rays.

Finally, the physician should not forget, if his patient be a young woman, that there may be hysterical spasm of the œsophagus.

Swelling and redness of the buccal mucous membrane occur in the various mild forms of stomatitis, and in the ulcerative type of this disease the more severe lesions are often found in this area. In the malignant ulcerative stomatitis called noma the slough which separates from the inside of the cheek leaves a large excavation which may become so deep as finally to perforate the cheek.

It is interesting to note that swelling of the cheek with great inflammation of the buccal mucous membrane is sometimes seen as the result of the formation of a salivary calculus in the duct of Steno, and it is also stated that obstruction from inflammation of this duct often occurs as a result of poisoning by sulphuric acid.

This writer has seen a case of that rare malady called Schönlein's disease, in which, in addition to the multiple arthritis, purpuric eruption, and great œdema, the formation of a large ulcer or slough threatened to perforate the cheek, and in healing produced a cicatrix which interfered with the patient's ability to open the mouth. This patient was an adult.

If a patient presents himself to the physician with the statement that he is suffering from general pains all over the body, particularly in the small of the back, quite high fever it may be, with much sore-throat and difficulty in swallowing, the trouble in the majority of cases will be, in the adult, tonsillitis of the follicular form. If the symptoms are exceedingly severe, the inflammation may result in suppuration—suppurative tonsillitis. It is to be remembered in all cases that the systemic or constitutional disturbance is out of all proportion to the severity of the local lesions. If it is tonsillitis, the glands can be felt in the majority of cases a little beneath and forward of the angle of the jaw, and pressure upon them may produce considerable pain. If the mouth is well opened and the tongue depressed, there will be found on each side of the throat a more or less projecting and inflamed mass, in the depressions or follicular openings of which will be found a white or yellowish exudate, which in severe cases may spread over the surface of the gland till it slightly resembles the membrane of diphtheria. Pressure on the tonsil may cause the further projection of these cheesy-looking masses.

In the suppurative form of the disease the surface of the gland may be smooth and reddened, and in a day or two become soft and fluctuating, and if lanced pus will escape.

The severe constitutional disturbance, the soreness of the throat, difficulty in swallowing, and the follicular exudate call to mind in all such cases the possibility of the disease being diphtheria; but in tonsillitis the exudate can be easily removed without leaving a bleeding surface behind it, and it has not the dusky, dirty look of diphtheritic membrane. Again, in tonsillitis the exudate is seen on the tonsils only, whereas in diphtheria it spreads over the half-arches and uvula. The general symptoms may make one suspect the onset of scarlet fever, particularly if the patient be a child; but the examination of the throat in scarlet fever shows the intense redness of the pharyngeal mucous membrane with comparatively slight enlargement of the tonsils. The intense redness of the throat in scarlatina and the development of the rash on the skin aid in making a differential diagnosis. The lymphatic glands of the neck may be enlarged in scarlet fever, but are rarely so in tonsillitis.

If the patient complains of dysphagia, and, on examination, the pharynx is red and the tonsils are covered with patches which speedily spread, as just described, so that by forty-eight or seventy-two hours the tonsils, pillars, and soft palate are covered by a gray membrane, the case should always be diagnosed as diphtheria and treated as such, unless a bacteriological examination of the exudate shows the infection to be due to a streptococcus and not to the Klebs-Loeffler bacillus. Even if the patient has not true diphtheria, he may be exceedingly ill. Again, it is to be remembered that, while many of the cases of scarlet fever which in their early stages present a membranous pharyngitis or tonsillitis are due to the streptococcus and not to the Loeffler bacillus, in the later stages of the disease the Loeffler bacillus is the cause of the local lesion. The differentiation is to be made chiefly by bacteriological tests, but it is worthy of note that the early formed streptococcic membrane does not spread as does the diphtheritic membrane, and does not return so rapidly when removed. The two diseases, diphtheria and scarlet fever, often exist simultaneously. Rarely the formation of a false membrane due to streptococcus infection, or still more rarely to the diphtheria bacillus, complicates the course of typhoid fever, and also occurs as a grave complication of measles.

If in any case of diphtheria the false membrane extends to the nasal chambers, the prognosis is very unfavorable.

Ordinary sore throat or acute pharyngitis is generally accompanied with little systemic disturbance, the local pain and soreness being the most characteristic symptoms. Inspection will show the pharyngeal wall red and angry looking, and very likely unduly dry. Care should always be taken, in the case of children particularly, that the early sore throat of measles and scarlet fever is not taken for simple pharyngitis. Often the rash of measles can be seen on the pharyngeal wall some hours before the rash appears on the skin.

Koplik asserts that this eruption on the pharyngeal mucous membrane is not characteristic. With this we cannot agree. He also asserts that an eruption appears on the buccal mucous membrane which precedes the eruption on the skin, and is characteristic before the skin eruption appears. It loses its peculiarities as the skin becomes involved and merges into a more diffuse redness, which entirely disappears before the skin has returned to its normal state. The buccal eruption consists of small, irregular red spots with a bluish-white centre, and should be looked for in a good light. Koplik believes that these spots are absolutely characteristic, and clinical experience endorses his view. (See Plate VIII.) Their absence, however, does not exclude the presence of measles. They are to be distinguished from the reddened mucous membrane of scarlet fever, the large, white spots of thrush, and the sore mouth of stomatitis. They do not appear in rötheln.

Pigmentation of the buccal mucous membrane often occurs in Addison's disease.

Sometimes cases are seen in which there are tonsillar pain and irritation, and in which careful examination proves the discomfort to be due to the presence of a small calculus in a follicle of the tonsil.

When swelling of the tonsils is chronic the enlargement of these bodies may produce mouth-breathing, with the peculiar facies of that habit (see illustration in chapter on Face), deficient thoracic and general systemic development, and a peculiar cough, constant in character and worse at night. Often the swollen or enlarged glands extending across the pharynx actually touch one another.

PLATE VIII.

Fig. I.

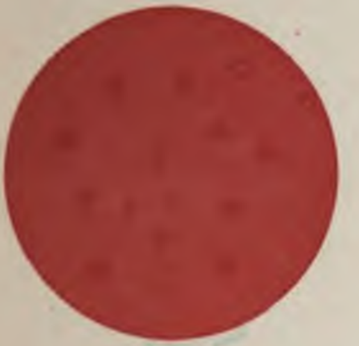


Fig. II.

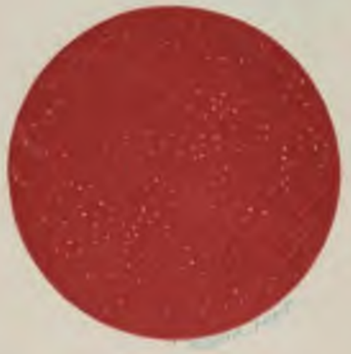


Fig. III.

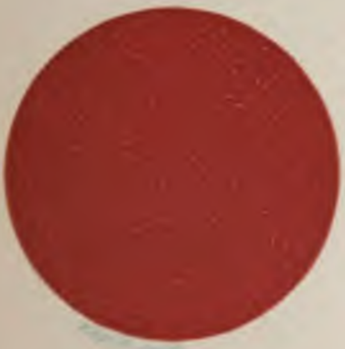
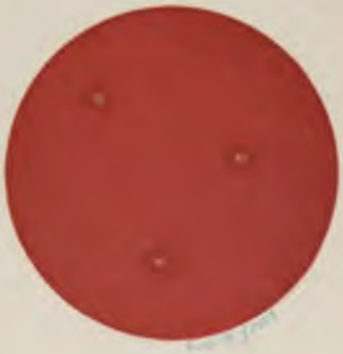


Fig. IV.



The Pathognomonic Sign of Measles
(Koplik's Picture of Flindt's Spots.)

Fig. 1.—The discrete measles spots on the buccal or labial mucous membrane, showing the isolated rose-red spot, with the minute bluish-white centre, on the normally colored mucous membrane.

Fig. 2.—Shows the partially diffuse eruption on the mucous membrane of the cheeks and lips; patches of pale pink interspersed among rose-red patches, the latter showing numerous pale bluish-white spots.

Fig. 3.—The appearance of the buccal or labial mucous membrane when the measles spots completely coalesce and give a diffuse redness, with the myriads of bluish-white specks. The exanthema on the skin is at this time generally fully developed.

Fig. 4.—Aphthous stomatitis apt to be mistaken for measles spots. Mucous membrane normal in line. Minute *yellow points* are surrounded by a red area. Always discrete.

CHAPTER VI.

THE SKIN.

The color of the skin—Eruptions on the skin—Gangrene, ulcers, and sloughs—Scars, sweating, dryness, œdema, hardness—Anæsthesia and hemianæsthesia—Paræsthesia, hyperæsthesia, itching.

MUCH information can be obtained by careful examination of the skin in many cases of disease. The examiner should make a note of the color of the integument, of its general nutrition, of its pliability, and of its sensibility. Naturally the eye at once takes in any eruption or scars which may mar its naturally smooth surface, and, as eruptions and scars are often the manifestations of more or less active systemic disorders, an insight into the presence of internal disease may be obtained from them.

The color of the skin in health in the white race depends upon the presence of pigment in the cells of the mucous layer of the epidermis, and in the corium in those parts of the body where pigmentation is marked, or to the condition of the subcutaneous circulation or of the blood in the subcutaneous vessels. Thus we often find the skin of the perineum, scrotum, axillæ, and of the lower abdomen much darker than elsewhere in persons in perfect health. Similarly we see a marked reddish or yellowish-brown hue in those parts of the skin which have been exposed to sun and weather, as a result of a deposition of pigment and an increased capillary circulation. With these normal alterations in color, however, we have little to do, for it is the abnormal colorations which interest us from a diagnostic stand-point. The most common of these changes in color due to pigment is jaundice; the next the chloasma of pregnancy or uterine disease, a condition usually limited to the face. Abdominal growths due to tuberculosis, cancer or lymphoma, and tuberculosis of the peritoneum also cause pigmentation of the skin, and in melanotic cancer there is often very dark discoloration, so marked as to be confused with that of Addison's disease. Again, it is not uncommon for persons who have hepatic torpor with constipation to develop what are called liver-spots, in which the skin has rather a dirty hue. Under the name of vagabond's "pigmentation" we sometimes see discoloration induced by the irritation of the skin produced by lice and exposure to dirt and weather, and this is capable of being mistaken for the pigmentation of Addison's disease. Finally, we see the yellowish-brown hue of the skin due to tinea versicolor, the bronzing of the skin in Addison's disease, and the slate-blue hue of argyria or chronic silver-poisoning. (See further on in this chapter.)

The changes in color depending upon disturbance of the subcutaneous circulation or on alterations in the blood are either local or general. In extreme nervousness flushing or blushing, due to a local vasomotor relaxation with increased blood-supply, may redden the face and neck, or in hectic fever a hyperæmia of the skin over the malar bones may give rise to an increase in color, which may be dusky red, due to imperfect oxidation of the blood. Considerable cyanosis of the face and hands in a case of tuberculosis of the lungs is a very grave symptom. Again, we see in pneumonia a peculiar dusky red flushing of one cheek or of the entire face, and in erysipelas the zone of hyperæmic redness is characterized by its sharp line of demarcation and its raised edge. In the alterations in color due to changes in the quality of the blood we have, as causes, anæmia due to lack of corpuscles or of hæmoglobin, arising from the various etiological factors producing such states.

JAUNDICE. Taking up the color-changes due to pigment, we find that in jaundice the deposition of the biliary coloring-matter varies in degree from a slight tinge or almost imperceptible yellowing to a dark citron or olive-green hue.

In examining the skin for jaundice care should be taken not to do so by gas- or candle-light, for the yellow flame masks the biliary color. If the tinge is very slight, it may be made more marked by stretching the skin on the palm of the hand or by pressing upon the skin a glass slide so that the yellow hue shows through it.

Having discovered that biliary coloring-matter has been deposited in the rete mucosum, it remains for the physician to decide what the cause of the jaundice may be. In the first place, it must be remembered that jaundice may be hepatogenous—that is, arise from disorder in the liver, or be hematogenous, from disorders of the blood with the setting free of blood-pigment. The hepatogenous jaundice is by far the more common of the two conditions, and the most common cause of this form of jaundice is catarrhal inflammation of the smaller ducts and common bile-duct which generally occurs in association with gastro-duodenal catarrh.

As a result of this catarrhal process the bile-duct becomes blocked by the swollen mucous membrane and the mucus which is secreted; the biliary coloring-matter is absorbed into the hepatic circulation and general circulation, and is by this means distributed over the body. Another common cause of hepatogenous jaundice is the obstruction offered to the flow of bile by the presence of a gallstone or gallstones in the ducts; and a third cause of obstructive jaundice, so called, is pressure on the ducts by growths or inflammatory products in the immediately adjacent organs, or of adherent inflammation in the ducts themselves, or by the presence of a round worm in the duct. Very rarely the jaundice may arise from the pressure on the common duct produced by floating kidney.

The following table, from Seymour Taylor's *Index of Medicine*, summarizes the causes of hepatogenous jaundice:

TABULAR VIEW OF THE CAUSES OF HEPATOGENOUS JAUNDICE.

Obstructive (feces clay-colored)	1. Gallstones and inspissated bile		In common duct. in radicles of duct.
	2. New growth	Malignant . . .	of liver itself. secondary infiltration of glands in transverse fissure. of stomach. of pylorus. of duodenum. of pancreas. of kidney.
		Non-malignant	syphilis. lymphadenoma.
	3. Catarrh of stomach and duodenum.		
	4. Abdominal aneurism.		
	5. Hydatid cysts.		
	6. Accumulation of feces.		
	7. Ovarian or uterine tumors.		
	8. Perihepatitis.		

Catarrhal jaundice of the acute type is generally produced by indiscretions in diet associated with exposure. The patient, after more or less marked symptoms of gastric and intestinal disturbance and indigestion, feels wretchedly. There is a premonitory mental heaviness, with languor and malaise, and within forty-eight hours or less the yellowing of the conjunctiva and skin appears. The temperature is generally subnormal to a slight degree. The tongue is heavily coated and often somewhat dry. There are marked loss of appetite, great distress, headache, and depression of spirits. Examination of the hypochondrium may reveal some local tenderness and slight hepatic enlargement, while the abdomen will be in some instances markedly tympanitic as a result of intestinal fermentative processes in the absence of antiseptic bile. The bowels are constipated, often refusing to move except with powerful purgatives. There is little pain, except headache. This condition lasts for a few days or a week, when the color of the skin and conjunctiva usually begins to fade and the normal hue is reached in the course of a week or more.

The presence of persistent jaundice should raise the suspicion that it is due to more serious disorder than simple catarrhal inflammation.

The jaundice from obstruction by stone may be due to blocking of the biliary duct, whereby there is a stagnation of the flow with reabsorption of the bile, or to stoppage of the flow by the presence of a stone in the common duct just as it enters the bowel. A differential diagnosis as to whether the stone is in one or the other of these places is usually impossible.

The jaundice of gallstone obstruction may be sudden or gradual in onset. If sudden, it is often, but not always, preceded by a violent attack of pain in the hypochondrium, or, in other words, hepatic colic, in which the agony is excruciating and is accompanied by nausea and vomiting. The area of the pain is, however, distinctly hepatic, and it does not radiate down the inside of the thigh and into the testicle or penis as does that due to renal calculus. In place of the subnormal temperature so often seen in catarrhal jaundice, we find in obstructive jaundice that the temperature is often considerably raised, and this is particularly apt to be the case in those instances in which the onset is gradual and the jaundice persistent, being due to reflex irritation or septic absorption produced by the impacted stone, which may be scratching or ulcerating the lining membrane of the duct. The history of repeated attacks of gallstone colic, the presence of gallstones now and then in the stools, the discovery of gallstone crepitus on deep palpation, the age of the patient, who is generally in or past middle life, and the fact that the patient is a female, all point to gallstone as a cause of the jaundice. As a rule, there is great loss of flesh in all forms of jaundice; but if the local damage done by the stone is great, and septic absorption is marked and the fever high, the failure in strength may be most alarming, while the repeated rigors and sweats increase the distress of the patient.

Jaundice very rarely arises from pressure on the ducts by an aneurism of the abdominal aorta, or from aneurism involving the hepatic artery. Three such cases are recorded by Frerichs. Jaundice has also been seen in aneurisms of the superior mesenteric artery as the result of pressure and in cases in which there has been, or is, perihepatitis, with displacement of the liver in such a way that the adhesions cause twisting or dragging on the ducts.

The juandice of malignant disease pressing upon the gall-ducts is usually not intense, and is characterized by the physical signs of a tumor, by the marked wasting of the patient, and, as a rule, by the very gradual onset of the pigmentation of the skin. Generally the lesion in such cases is carcinoma of the head of the pancreas.

In connection with this possibility the law of Courvoisier is to be borne in mind. This law has been well expressed by Mayo Robson in the following words: "Jaundice with distended gall-bladder is presumptive evidence in favor of malignant disease, but jaundice without distended gall-bladder favors the diagnosis of cholelithiasis." While this law or rule is a good guide, it must not be forgotten, as pointed out by Kehr, that gallstones produce malignant disease. Thus Courvoisier found gallstones in 87.5 per cent. of malignant cases, Delano Ames in 95.4 per cent. of gall-bladder cancer, and Schroeder reached similar conclusions.

Jaundice is also seen in hepatic hypertrophic cirrhosis to a slight extent in a small proportion of cases, and it is to be remembered that in those cases of this disease in which delirium and muscular twitching occur the symptoms may resemble acute yellow atrophy of the liver, and that all forms of jaundice produce headache and may cause delirium. In acute yellow atrophy of the liver (see below) the liver is greatly reduced in size, whereas in hypertrophy it is greatly increased in size; and in atrophy the temperature is subnormal, whereas in the jaundice due to hypertrophic cirrhosis it is apt to be above normal. Jaundice also may be a manifestation of acute poisoning by phosphorus, which condition is generally accompanied by hepatic swelling and tenderness and with coffee-ground vomiting.

Jaundice is present in all fatal cases of yellow fever and often in cases which ultimately recover. It also is a constant symptom in Weil's disease, which is probably in reality a septic icterus, but it is very rarely seen in suppurative hepatitis. A fleeting and light hue of jaundice is sometimes seen in cases of chronic valvular cardiac disease in which compensation is gradually failing. Rarely this hue becomes deeper as the heart-failure increases. This jaundice is due to engorgement of the liver (nutmeg-liver), which in time results in catarrh of the bile-ducts, with consequent obstruction to the flow of bile.

In amyloid disease of the liver Bartholow states that jaundice occurs in about one-tenth of the cases as a result of enlargement of the lymphatics in the hilus with pressure on the hepatic duct. In jaundice resulting from cancer of the liver the growth must be so situated as to compress the ducts, consequently jaundice occurs in only about one-third of the cases. Similarly jaundice may result from the presence of echinococci, but this is not a common symptom of the growth of these parasites, and the disease is very rare in the United States.

The association of gastric disorder with evidences of gastric dilatation and jaundice should recall the fact that gastroptosis sometimes causes jaundice.

Jaundice sometimes complicates diabetes. Under these circumstances it may be regarded as a coincidence or a valuable diagnostic aid, for, as we have already stated, tumors of the pancreas by pressing on the common duct may cause jaundice, and, as is now well known, widespread disease of the pancreas may cause diabetes. Jaundice in a case of diabetes should, therefore, direct attention to the pancreas.

In this connection it is well to remember that Hanot, under the name of diabète bronzé, has described a pigmentation of the skin which contains iron (that of Addison's disease and melanæmia does not), and which is associated with diabetes, hypertrophic cirrhosis of the liver, and enlargement of the spleen. The coloration occurs most markedly upon the face, limbs, and genital organs; the glycosuria is abundant and slight ascites may be present, the lower limbs may be œdematous, the loss of weight and strength is rapid, and death soon ensues from pneumonia or coma. Hanot and Marie both regard it as a disease distinct from ordinary diabetes mellitus.

True diabète bronzé is exceedingly rare. Up to 1897 only thirty cases had been reported. In the four cases reported in America it is interesting to note that diabetes was not present. It is probably better to call the disease "hepatic cirrhosis with pigmentation."

Other noteworthy symptoms of hepatogenous jaundice are intense itching of the skin; a very slow pulse when the patient is at rest, due to stimulation of the vagus by the bile in the blood; and staining of the sweat due to the bile-pigment may also be present. Should the jaundice be due to gallstones impacted in the ducts, and producing irritation or ulceration of their lining so that septic absorption or "Charcot's fever" develops, the pulse may become more rapid and running, from the general feebleness which rapidly asserts itself. Rigors of extreme severity, followed by sweatings and marked febrile movement, develop in such cases, the chills occurring daily or periodically in a manner closely resembling those of intermittent fever. As these symptoms sometimes develop in cases in which the post-mortem discovers no sign of pus, it has been thought that the disturbances were due to reflex causes; but the opinion of Charcot that there is present in all such cases a true infection seems the more probable. When the gallstone produces active suppuration the fever becomes more like remittent fever and the patient rapidly emaciates and presents all the signs of active suppuration.

The urine in all cases of hepatogenous jaundice is heavily bile-stained (see Urine), and the stools are generally clay-colored owing to absence of bile in the feces.

A very rare cause of jaundice is acute yellow atrophy of the liver, a disease which is seen somewhat more frequently in women than in men, and particularly in association with pregnancy. The age of occurrence is usually between the twentieth and thirtieth years. The symptoms begin with gastro-intestinal disorder, followed by headache, delirium, muscular twitching, and perhaps convulsions. Simultaneously with the onset of the headache the jaundice appears, the patient becomes typhoidal and dies from exhaustion, although recovery has been known to occur. The stools during the attack are clay-colored, and the urine contains leucin in disks and tyrosin in needle-like crystals.

Hematogenous jaundice is due, as its name implies, to breaking down of the blood to so great an extent that the liver cannot deal with the waste material with sufficient rapidity, and as a result altered hæmoglobin is deposited in the tissues. Any poison which produces excessive *hemolysis*, such as picric acid and the coal-tar products, chlorate of potassium, glycerin, and poisonous mushrooms, may cause this condition to develop, and in extreme malarial disease (remittent and pernicious malarial fever), dengue, relapsing fever, pernicious anæmia, pneumonia, and in other infectious maladies jaundice may be produced in this manner. It is particularly apt to occur in cases of marked sepsis.

Its causes are shown in the following table, from Seymour Taylor's *Index:*

CAUSES OF HEMATOGENOUS JAUNDICE.

- Non-obstructive (feces normal in color)
  - Fevers . . .
    - Yellow fever.
    - Typhus fever.
    - Scarlet fever.
    - Relapsing fever.
  - Poisons. . .
    - Animal
      - Snake bite.
      - Pyæmia.
    - Chemical
      - Phosphorus, copper, mercury, antimony, chloroform, ether.
  - Acute atrophy of liver.
  - Neurosis: joy, grief, fear, passion.
  - Cirrhosis of liver in its later stages.

Jaundice sometimes occurs after severe hemorrhage of a prolonged character and in prolonged exhausting fevers, and is then due not to any local hepatic trouble, but to blood-changes, with the production of urobilin in excessive amounts. The urine fails to carry off all the urobilin which is produced from hæmatoidin or bilirubin. This condition is called "urobilin icterus."

In nearly all cases of hematogenous jaundice the discoloration of the skin is very slight, and the important fact is to be remembered that the stools are not light or clay-colored as in hepatic jaundice, but contain a normal or excessive amount of pigment. Again, the systemic symptoms of catarrhal or obstructive hepatic jaundice are practically absent in the hematogenous variety, and the juandice is simply a minor symptom associated with more grave manifestations which characterize the individual infectious process. If the poisoning is very marked, convulsions, coma, or active delirium may come on, but it is probable that these symptoms are due more to the poison of the disease than to the broken-down blood.

Vierordt states that a very small amount of biliary coloring-matter is often found in the urine of patients suffering from pyæmic jaundice, and regards this as an important sign that the discoloration of the skin is due in a given case to blood-changes and not to biliary obstruction, whereas an excessive amount of biliary matter in the urine indicates hepatic trouble.

Jaundice sometimes complicates croupous pneumonia, and if hematogenous in origin is a most serious condition, for it usually indicates a fatal ending. When such an attack arises from catarrh of the gall-ducts during an attack of pneumonia the condition is by no means so grave.

There remains to be considered the jaundice seen in the newborn, usually within the first or second day of life (icterus neonatorum), which some believe to be due to a decrease in the blood-pressure in the portal vessels subsequent to the arrest of the placental circulation, with consequent absorption of bile into the blood, owing to the comparatively high tension of this fluid in the bile capillaries. Others think this jaundice is due to breaking down of the blood-corpuscles shortly after birth as the result of some mild infection. Probably both causes act in some cases. If the cause be altered blood-pressure, the prognosis is favorable, and recovery takes place in about ten days or two weeks; but if the cause be an infection, the condition often proves rapidly fatal. Should this jaundice of the newborn be very marked the patient may be suffering from congenital stenosis, or absence of the common or hepatic duct (which cause is rare); from septicæmia, through infection by way of the umbilicus; from phlebitis of the umbilical vein, or from a hepatitis due to hereditary syphilis. In any of these latter causes death will probably occur, whereas in the mild form of icterus neonatorum the prognosis is very favorable, even though the discoloration lasts for weeks. The mild form of icterus neonatorum, if due to blood-changes, is rarely accompanied by great discoloration of the urine, and the feces are usually no lighter than normal in color; but if hepatic disease be present, the urine is bile-stained and the feces are light in hue.

Jaundice sometimes comes on in the course of acute ulcerative endocarditis, and has been mistaken for that of acute yellow atrophy of the liver, and it often appears as a symptom of pernicious malarial fever, with vomiting, diarrhœa, and grave nervous symptoms.

Rarely jaundice follows severe fright or extreme anger, and Da Costa states that it sometimes ensues after concussion of the brain.

OTHER CHANGES IN THE COLOR OF THE SKIN. A condition of the skin characterized by yellow, more or less elevated patches, is xanthoma, which Murchison states often complicates hepatic disease, but in the writer's experience it is usually met with in gouty women or patients suffering from oxaluria. Its favorite distribution is about the eyelids, but it may appear elsewhere. Lesions similar to xanthoma sometimes appear in the course of diabetes (Hutchinson, Besnier), and under these circumstances generally develop suddenly, and spontaneously disappear after some weeks or months.

When the skin of the entire body, the face being particularly affected, is of a livid or bluish-slate color, resembling somewhat the appearance of a person exposed to rays of light passing through blue glass, the condition is that of argyria or chronic silver-poisoning. This discoloration is so characteristic as to admit of no difficulty in diagnosis, since the absence of any circulatory or respiratory embarrassment excludes the possibility of its being due to cyanosis. Owing to the small amount of silver now given internally by physicians, chronic argyria is becoming more and more rare. The discoloration is due to a deposit of oxide of silver in the rete Malpighii.

Discoloration of the skin of the entire body of a sallow, lemon-yellow tint, sometimes called a "muddy-yellow" hue, is seen in persons who are sufferers from prolonged malarial poisoning, and in some cases the subjects of prolonged suppurative processes not tubercular in character. A greasy, yellowish skin does, however, occur as an accompaniment of some cases of pulmonary phthisis, and these cases have, as a rule, a gloomy prognosis. Often chronic hepatic disease, such as cirrhosis, produces this sallow appearance.

Other changes in the color of the skin, which cannot be said to be due to deposition of pigment, although they seem to be caused by this, are seen most markedly in the peculiar yellowish, cheesy pallor of carcinoma, the greenish-yellow tinge of true chlorosis, the curious cadaveric hue of advanced pyæmia, and the yellow skin with a greasy feeling in some cases of paretic dementia.

Local pigmentation of the skin results from many causes, both local and systemic, direct and indirect. When brownish-yellow spots or streaks appear on the face, so that chloasma is developed, we should look for uterine or hepatic disturbance or pregnancy; they are practically large freckles of a more or less distinct brown hue. Sometimes on the skin of the trunk small yellowish-brown or chamois-skin colored spots appear accompanied by no other symptoms except perhaps slight itching. This is due to phthiriasis versicolor or chromophytosis due to the growth of the parasite microsporon furfur. The diagnosis can be settled by painting the infected area with Lugol's iodine solution, when the spot becomes a dark-brown or mahogany color. This is called "Allen's Test." In other instances chloasmic spots or localized discoloration of the skin results from injury to the skin, as pressure by clothes, chafing, or after constant severe scratching in the course of eczema or pediculosis or scabies. If the pigment is found in the nuchal and sacral regions, it is probably from the scratching caused by pediculi; if on the body in irregular distribution, it may have been caused by prurigo. Again, the presence of a brown pigmentation of the skin in clearly outlined patches may indicate the earlier use of a fly-blister, a mustard plaster, or other counter-irritants, and a brown discoloration of the skin, which might possibly be confused with that of Addison's disease, is produced by the free use externally of oil of cade. Sometimes these spots are produced by the prolonged use of arsenic, and the writer has reported a case in which the coalescence of the spots produced a curious grayish-brown hue of the entire body, so that the man looked somewhat like a mulatto.

Sometimes brown pigmentation of the skin of the neck and face appears as a symptom in exophthalmic goitre, and this disease may also produce similar lesions on the chest and wrists.

Very closely resembling these spots is the bronzing of the skin in patches which is seen in persons suffering from Addison's disease; but although bronzing of the skin is a somewhat constant symptom of Addison's disease, its presence is neither a positive nor negative sign in diagnosis, for bronzing is sometimes seen in cases in which the suprarenal capsules are normal. In some instances the bronze color deepens into a dark gray or even a black hue, and although the discoloration is generally in patches, it may extend over the entire surface of the skin, even to the edges of the finger-nails. The nails, however, escape, as does also the mucous membrane of the lips, although the lining of the mouth itself may be dotted with pigmentation. The color is due to pigmentation of the rete Malpighii, and pressure has no effect on it. The symptoms of Addison's disease to be found associated with these skin-changes are "anæmia, general languor or debility, remarkable feebleness of the heart's action, and irritability of the stomach." (Addison.)

The slate-colored skin of argyria or chronic silver-poisoning can be readily distinguished from the bronze color of Addison's disease; but if a further test is needed, it will be found that washing the skin of argyria with a solution of iodine changes its color, while that of Addison's disease remains unaltered.

White patches, or leucoderma, are also sometimes seen in cases of true goitre, and brown ones in tuberculosis.

In carcinoma of one of the internal organs, or of the breast of an advanced stage, the appearance of the skin is drawn and unusually smooth, often shiny or greasy-looking, somewhat gummy and leathery to the touch, particularly where the integument is naturally dense. Although it is difficult to describe, this skin is almost pathognomonic of carcinoma, although it may also be present to some extent in far-advanced cases of pernicious anæmia or sarcoma.

Pallor of the skin is due to absence of the normal pigment, to deficient blood, to central or local vasomotor disturbance as is typified by fainting, and far more rarely by Raynaud's disease. As a type of the pallor due to lack of pigment in the skin we see vitiligo, while the pallor due to pernicious anæmia or pseudo-leukæmia and malaria is owing to lack of red corpuscles. Similarly, a pallor due to lack of hæmoglobin is typified by chlorosis. (See Blood.) In all of these diseases the skin may be of ghastly whiteness or tinged with yellow. The skin is apt also to be very white, and even chalky in appearance, in chronic contracted kidney and chronic parenchymatous nephritis.

In chlorosis the entire surface of the body is exceedingly pale, and the skin of the face, particularly about the mouth and nose and eyes, is somewhat greenish in hue.

A very important diagnostic point to be remembered is that red cheeks often cause the physician to overlook well-advanced anæmia in young women. (See chapter on the Blood.)

In those cases in which the skin is pale from alteration of the subcutaneous circulation there is usually incompetence of the heart or vasomotor disturbance, but the most marked form of general pallor is that due to myxœdema.

Cyanosis, or blueness of the skin, depends upon the circulation in the subcutaneous vessels of imperfectly oxidized blood. The small veins are often seen to be swollen, particularly those of the face and the hands and feet. The most marked form of cyanosis with which we meet is the cyanosis of the newborn child, suffering from a patulous foramen ovale, and in this condition the color may vary from a slate-hue to an almost black hue. The lobes of the ears, the tongue, the scrotum, and the toes show the color most deeply. It is important to remember that this form of cyanosis is greatly decreased, as a rule, by placing the child on its right side. Anything which produces excitement increases the cyanosis greatly, whereas cyanosis due to other causes is not subject to great variations. In the cyanosis of the newly born, males are far more frequently affected than females, in the proportion of about 2 to 1 or 3 to 1, and it is a noteworthy fact that even when the cyanosis is due to a malformation of the heart it may not be present from the time of birth, but may develop several days afterward. J. Lewis Smith records forty-one cases in which the cyanosis due to congenital heart-lesion came on at periods ranging from two weeks to forty years after birth.

About 35 per cent. of the cases of cyanosis due to congenital defects die in the first year. The following table, from J. Lewis Smith, shows the character and relative frequency of these lesions:

	Cases.
1. Pulmonary artery absent, rudimentary, impervious, or partially obstructed	97
2. Right auriculo-ventricular orifice impervious or contracted	5
3. Orifice of the pulmonary artery and the right auriculo-ventricular aperture impervious or contracted	6
4. Right ventricle divided into two cavities by a supernumerary septum	11
5. One auricle and one ventricle	12
6. Two auricles and one ventricle	4
7. A single auriculo-ventricular opening; interauricular and interventricular septum incomplete	1
8. Mitral orifice closed or contracted	3
9. Aorta absent, rudimentary, impervious, or partially obstructed	3
10. Aortic and the left auriculo-ventricular orifice impervious or contracted	1
11. Aorta and pulmonary artery transposed	14
12. The cavæ entering the left auricle	1
13. Pulmonary veins opening into the right auricle or into the cavæ or azygos veins	2
14. Aorta impervious or contracted above its point of union with the ductus arteriosus; pulmonary artery wholly or in part supplying blood to the descending aorta through the ductus arteriosus	2
Total	162

The chances are about ten to one that in cyanosis of the newborn the lesion is absence of a properly developed interauricular or interventricular wall.

In the adult or child cyanosis may be produced by serious cardiac disease, by pulmonary disease, such as pneumonia, pulmonary congestion, and bronchiectasis with emphysema and associated cardiac dilatation. It also occurs in laryngeal obstruction arising from external pressure or intralaryngeal difficulty, and in cases of asthma of a severe form. (See chapter on the Thorax and its Viscera.)

In some cases of paretic dementia the skin of the forehead is dull and dusk-looking. In other instances a grayish-blue or cyanotic appearance may arise from the ingestion of drugs which reduce the hæmoglobin of the blood, such as antipyrine or acetanilid, and in such instances the discoloration is first seen about the base of the thumb-nail or in the skin of the face, particularly if the patient be examined from a little distance.

The condition of the skin, so far as its nutrition is concerned, is of great importance in diagnosis. In profound failure of the vital forces continuing over a great length of time it becomes abnormally dry and scaly, the hair becomes straggling and lustreless, and frequently falls. In young persons suffering from grave disease of the lungs or heart of a chronic type there is often not only an undue dryness of the cuticle, but an abnormal growth of downy hair all over the body and limbs, and more particularly down the spine and over the breast-bone.

Eruptions on the Skin. The influence of age upon the development of skin lesions is very great, and Stephen Mackenzie has summed up the relationship of skin diseases to age in the following amusing manner: "The seven stages of man could be well illustrated by disease of the skin, though we lack a Shakespeare to do justice to the theme. In the 'mewling and puking' infant we meet with sclerema and œdema neonatorum, the 'red gum' or strophulus of the older writers, intertrigo, eczema, urticaria papillosa (lichen urticatus), urticaria pigmentosa, xeroderma pigmentosum, and impetigo; the 'schoolboy,' with his chilblains and ringworms, alopecia areata, pityriasis rosea, ecthyma, and 'football disease;' and then the 'lover,' with his acne and sycosis, and, as a result of irregular sexual excursions, his syphilides; 'and then the justice, in fair round belly,' with acne rosacea, diabetic boils, and pruritus ani; the sixth stage shifts into the 'lean and slippered pantaloon,' with rodent ulcer and 'gouty' eczema; 'last scene of all, sans teeth, sans eyes, sans taste, sans everything'—except an incessant and intolerable itching of the skin which we call senile prurigo."

There are two conditions of the skin in which valuable evidence is given that the patient is suffering from rheumatism. One is the presence of erythema in one of its many forms, the other is the appearance of purpura, or, as it has been called, peliosis rheumatica. That the presence of erythema points in many cases to rheumatic trouble is proved beyond all doubt, either erythema papulatum, annulaire, marginatum, or nodosum being indicative of the systemic taint; but it is worthy of note that the erythema marginatum is most diagnostic and erythema nodosum the least diagnostic of rheumatic poisoning. Sometimes this eruption may be the only manifestation other than cardiac involvement, and when the marginate eruption is present severe cardiac involvement is commonly seen. The papulate eruption is most commonly found on the back of the wrists, the hands, and the feet when it occurs as a rheumatic sign, while the nodose variety is generally confined to the front aspect of the legs or the extensor surfaces of the arms. It must be remembered that these forms of erythema may be distributed anywhere over the body in rheumatism, but that they become especially diagnostic if limited to the areas named.

Purpuric discolorations of the skin, somewhat resembling multiple bruises in appearance, are due to a number of causes and possess a varied significance. In the first place, they are due to the disease known as purpura hæmorrhagica, which may be divided into the acute and subacute forms and that which is secondary as the result of severe infections and certain poisonings. The acute form of purpura runs a rapid course and reaches a fatal result in most cases in a short time. It is a comparatively rare disease and usually attacks young adults, chiefly males, up to twenty-eight years of age. It is sometimes seen in young girls and more rarely in young pregnant women. The chief symptoms consist in hemorrhages from the mucous membrane, purpuric spots, high fever, and a general class of symptoms resembling those of sepsis, as chills, pyrexia, and exhaustion. In other instances active hemorrhages take place into the viscera, and if into the meninges of the brain cause cerebral symptoms at once. The liver and spleen are nearly always enlarged.

The subacute type, while severe, runs a far more favorable course as to its manifestations and results. It usually attacks children or young adult males from twenty to thirty years of age. The patient, after a feeling of wretchedness, and perhaps a chill, followed by the purpuric eruption, is attacked by swelling of the joints and perhaps hemorrhages from the kidneys, bowels, and mucous membranes. If the hemorrhage be from the gums, the teeth are not loosened, as in scurvy. Prostration may be great and the patient may appear as if suffering from typhoid fever. The prognosis is good for ultimate recovery. It is sometimes called peliosis rheumatica or Schönlein's disease. This subacute form, however, occurs in a more severe manner, as "Henoch's disease," in the persons of children between nine and twelve years, and is much more common in males than females (five to one). In this form we have as additional symptoms marked pain and tenderness in the belly and bloody stools, with tenesmus and active vomiting. The illness may last a long time, but recovery often occurs, about 25 per cent. dying. The joint-symptoms of the other forms of purpura may be slight or absent. Often, too, the purpura is accompanied or replaced by erythema.

The question as to whether purpuric eruptions are ever truly indicative of rheumatism has been much discussed and their diagnostic value denied, but the author believes that in some cases of rheumatism purpura is a symptom, appearing often in the neighborhood of the involved joints, nearly always on the lower limbs, and often breaking out before any evidence of articular trouble exists. In other instances the development of the purpura is simultaneous with the disappearance of joint-trouble. The eruption usually fades in a few days, but frequent relapses or new crops of it often occur.

Purpuric eruptions may be produced by quinine in persons who have an idiosyncrasy to this drug, and by iodide of potassium, chloral, and salicylic acid. They may also accompany any severe infectious disease and follow the entrance into the body of any poison which destroys the blood, such as the poison of snake-bite. They also result from severe jaundice, from profound anæmia, from congenital syphilis with vascular changes, from endocarditis (a form of sepsis), and in cases of multiple sarcomata. Rarely purpura has followed fright and severe grief.

Hemorrhages of the skin occur spontaneously in some cases of hysteria and paretic dementia, and after epileptic attacks, particularly about the eyes, and often from injuries received in other parts of the body during the convulsion. Minute hemorrhages may also occur in the course of severe whooping-cough, and, in the form of petechiæ, result from snake-poisoning, septicæmia, cerebro-spinal meningitis, iodism, ergotism, and after inhaling the vapor of benzine. They are also seen in scurvy and in some cases of profound wasting, as in the course of phthisis and carcinoma.

Petechial rashes closely resembling those of malignant smallpox, typhoid fever, or cerebro-spinal fever may be due to the presence of acute ulcerative endocarditis.

Hemorrhages sometimes appear in the skin covering a part which has been affected by a severe pain in a crisis of locomotor ataxia.

A very extraordinary manifestation of spontaneous subcutaneous hemorrhage is seen in what is known as hæmatoma auris, a condition in which a free extravasation of blood takes place into and beneath the skin of the ear. The color of the swollen ear is quite red, if the hemorrhage has been recent, or dark blue if it is an old occurrence. The left ear is more commonly affected than the right, and it is seen more commonly in males than females.

The development of polymorphic skin lesions, consisting of hyperæmia, œdema, and hemorrhage, with arthritis occasionally and visceral disturbances, consisting in attacks of vomiting or diarrhœa, endocarditis, pericarditis, acute nephritis, and hemorrhages from the mucous membranes, indicates the presence of a condition called erythema exudativum multiforme. The attacks are apt to be recurrent. Sometimes the skin-manifestations are absent.

Subcutaneous fibroid nodules sometimes occur in cases of rheumatism and vary in size from a hemp-seed to a walnut. They are usually situated in the subcutaneous connective tissue, but may be attached to the deep fascia or muscular sheaths.

Intense redness of the skin is seen in acute inflammations of the skin or the subcutaneous tissues, and as the result of hot applications, the redness being more and more marked as the heat is prolonged and is great. Often the prolonged use of high heat will produce a peculiar mottling of the skin like that of an old bruise.

Aside from the redness of the cheeks and forehead from blushing, we should remember the general flushing seen so commonly in persons suffering from phthisis, particularly when they are excited, which differs from the more dusky redness seen over the malar bones in hectic fever.

Urticaria may occur as a manifestation of rheumatism, but it has no diagnostic value. Sometimes it ensues upon the use of salicylic acid or turpentine, and quite commonly follows the ingestion of iodide of potassium. The wheals produced by the latter drug differ from those of urticaria in being unduly red.

Another interesting diagnostic sign in the skin is what is known as the "*tache cerebrale*," a condition of vasomotor disorder in which when the finger is gently drawn over the skin of the forehead a red patch speedily develops. It is seen in meningeal irritation, brain abscess, epilepsy, in some cases of exophthalmic goitre, and in paretic dementia. Sometimes it is called "*tache meningeale*."

Erythema or rose-rash, sometimes called roseola, is a redness of the skin, and occurs in many pathological conditions. It may be localized or diffused. In a number of diseases it aids us very greatly in reaching a diagnosis, but the physician should always be cautious in depending much upon it, since it may mislead, owing to the fact that it often appears when devoid of diagnostic importance.

The development of a diffuse, punctated rose-rash on the skin of a person who is suffering from malaise, fever, nervous disturbance, and sore throat should direct the physician's attention to the possible presence of two infectious diseases, namely, scarlet fever, which is more common in childhood, and syphilis, which is more frequent in adults. The rash of scarlet fever is usually of a bright-red color, and shows itself at the end of the first or on the second day of the disease, first appears on the chest and neck, and then speedily involves nearly the whole surface of the body, although the forehead often escapes and the skin about the corners of the mouth remains nearly always white and free from eruption. On the other hand, the soles of the feet and palms of the hands are very markedly affected. So intensely reddened is the patient's surface that it may have the color of a boiled lobster. This redness depends upon an acute hyperæmia of the skin, which though removed by pressure instantly returns when the finger is withdrawn. A noteworthy point is its punctate and mottled appearance, for, while the entire skin may be red, there are points which are more red than the rest of the skin, and also certain areas which are particularly so. The skin is often slightly swollen and feels tense, and itching is commonly present. The rash usually lasts three or four days, and then fades, desquamation of the cuticle speedily setting in, which is complete in about two weeks. Sometimes, however, it remains for ten days to three weeks. Often when the rash can scarcely be seen on the skin its full development will be found on the pharyngeal wall. In the malignant types of scarlet fever petechiæ and subdermal hemorrhages occur. Sometimes in scarlet fever fine miliary vesicles develop chiefly in the neighborhood of the axillæ or on the abdominal wall or thorax anteriorly.

Sometimes, too, in those cases of scarlet fever which have severe symptoms of ulcerating sore throat with ear or nose complications there develops, about the third week of the disease, a dark-red papular or macular erythema on the extensor aspects of the large joints. It is a grave symptom.

An erythema resembling scarlet fever, not only in its appearance, but also by its association with swelling of the lymphatic glands and reddening of the mucous membranes of the mouth, sometimes develops about the second or third day in cases of dengue or breakbone fever.

In children there are several other conditions than scarlet fever which are associated with rose-rash, and these are more apt to lead to errors in diagnosis than is the rash of syphilis. The most frequent of these is erythema roseola, or roseola of acute indigestion, or that following the use of a food to which the patient has an idiosyncrasy. It is generally, but not always, widely diffused and is often associated with acute and severe febrile movement and vomiting, but it can be separated from scarlet fever by the facts that there is an absence of severe constitutional and nervous symptoms (except in neurotic children), there is no sore throat or enlarged cervical glands, and the rash does not come out on the clavicles and gradually travel down the body. Roseolous eruptions also appear in persons with delicate skins after coming in contact with irritant plants, and Dukes asserts that they may develop from handling caterpillars.

Dukes has also described a febrile condition closely resembling rubella or rose-rash and scarlet fever, which he believes to be a separate entity, namely, what he calls the "fourth disease." It is a mild infectious complaint, less severe than scarlet fever and more severe than rubella, the temperature, as a rule, is moderate, and there is little feeling of illness. The disease runs a very short course, and the lymphatic glands are markedly and widely affected, though not as much so as in rötheln. The points on which Dukes bases his diagnosis of the "fourth disease" are its prolonged period of incubation (nine to twenty-one days); the usual absence of pre-eruptive vomiting, the low pulse-rate, the absence of desquamation of the tongue about the fourth day, and the absence of renal complications. Many physicians believe that this condition is really a very mild scarlatina or rubella, and grave doubt is felt by nearly all observers as to its being a separate entity.

Another condition closely resembling scarlet fever is rarely seen, namely, acute exfoliating dermatitis, called, in its mild form, *erythema scarlatiniform*, which has a sudden onset with febrile movement and a rash which rapidly spreads over the entire body and lasts four or five days, finally ending in desquamation. So closely may this disease resemble scarlet fever that a diagnosis during the first attack may be impossible for the first few days, but the condition of the throat and tongue does not resemble the condition seen in scarlatina. Desquamation is often even more complete than in scarlatina, and the hair and nails are frequently shed. Relapses are very common and give rise to the reported cases of repeated attacks of scarlet fever.

The rash of rubella or rubeola or rötheln (German measles) is a roseola, and more closely resembles that of scarlet fever in some cases than it does that of measles, but it is never as scarlet, is distinctly maculated, and only at a distance looks homogeneous. Like measles, it is first seen on the face, chiefly about the nose and on the upper lip. Close examination always reveals the rash in oval patches or crescents, and it lacks the diffused character of the rash, the punctation of the skin, the grave systemic disturbance, and the throat-symptoms of scarlet fever. Further, the febrile movement is comparatively slight, and the rash lasts only twelve hours or for two or three days. Slight desquamation may, however, occur.

Rötheln, or German measles, is separated from true measles in many cases by the marked glandular enlargements, chiefly the posterior cervical, axillary, and inguinal; but this is also constantly true of scarlet fever and sometimes of measles. It never presents "Koplik's spots" on the buccal mucous membrane. (See chapter on Tongue.)

The eruption of measles (morbilli) is very characteristic, and can be in most cases easily separated from the other exanthemata by close examination. It is a roseola in character, but more dusky than that of scarlet fever. It appears about the fourth day of the illness in association with catarrh of the mucous membrane of the eyes and respiratory tract. Unlike scarlet fever it appears in macules first upon the forehead or face, then on the neck, trunk, and limbs. The macules, which often coalesce, are arranged in crescents which are red, but become somewhat yellowish on pressure. They are slightly raised. There is nearly always to be seen some uninvolved skin, the entire surface not being covered as in scarlet fever. In some instances in which the eruption is aberrant a diagnosis of measles from scarlet fever is admittedly impossible until the case has been watched for some days; but the slow onset of measles, in which the eruption appears on the fourth day as against the first day in scarlet fever, the swollen eyes and nose, the puffiness of the face, the catarrhal condition of the mucous membranes, the curious fall of temperature after the preliminary rise on the first day, the short duration of the rash, all aid in the diagnosis of measles. The dusky eruption of measles can nearly always be found on the pharyngeal mucous membrane. (For the mouth and throat symptoms of scarlet fever and measles, see chapter on the Mouth and Tongue.)

An erythema or roseola sometimes appears over the skin of children who have been vaccinated, generally about eight to ten days after the operation. It rarely lasts more than two days, and on its disappearance there is slight desquamation.

It also appears sometimes in cases of smallpox previous to the outbreak of the true eruption. Under the latter circumstance it is found most commonly about the groin and inner surface of the thighs and on the hypogastrium, loins, clavicles, and the extensor surfaces. So closely may the early rash of smallpox simulate the aberrant type of measles as to lead to grave mistakes in diagnosis. Sometimes an immediate diagnosis is impossible, even by the most experienced, but the rash of measles commonly appears on the face, therefore this difference, coupled with a history of exposure, the gradual development of the peculiar "shot under the skin" sensation of variola, and the ultimate distinct papulation, vesiculation, and pustulation of smallpox soon remove the doubt from the physician's mind.

DAY OF ERUPTION OF THE VARIOUS EXANTHEMATA.

Day.	Disease.	Area.
First to second day	Rötheln or German measles. Varicella or chickenpox.	Face first. Face or trunk.
Second day	Scarlet fever.	Neck and chest
Third to fourth day	Measles or morbilli or rubeola. Variola or smallpox.	Face. Forehead, face, and wrists.
Fourth to fifth day.	Typhus or ship fever.	Trunk.
Seventh to ninth day	Typhoid or enteric fever.	Abdomen.

The remembrance that the incubation period of variola is twelve days, that of varicella seventeen days, of measles ten days, of rubella twenty-one days and of scarlet fever two to four days, will aid the diagnosis if a history of exposure can be obtained.

A diagnosis between the eruption of measles and variola often can be made by stretching the skin between the fingers, when, if it be measles, the papule cannot be felt, whereas, if it be variola, it persists. This is called the "grisolle sign."

Among other diseases in which rose-rash appears we find diphtheria, septicæmia, cholera, typhoid fever, malarial poisoning, and Bright's disease. In diphtheria it may lead the physician to a diagnosis of scarlet fever with severe faucial manifestations, and only a careful examination of the throat, the rapid subsidence of the rash, and the bacteriological examination of the false membrane will settle the diagnosis. Sometimes, however, a roseola appears late in the course of diphtheria, probably as a result of septic absorption. In other instances the injection of anti-diphtheritic serum or other antitoxins produces this result. The presence of a very high temperature, of nervous irritability, and the predominance of the throat-lesions of scarlet fever ought to decide the diagnosis in favor of scarlet fever.

The physician should also recall the fact that the injection of anti-diphtheritic serum sometimes causes a roseolous eruption, associated, it may be, with pains in the joints. The general illness caused by diphtheria, plus these symptoms, may point to a complicating scarlet fever or measles. The antitoxin rash is not, as a rule, so persistent as that of scarlet fever, lasts a short time, and is rarely followed by desquamation, except in fine scales. While it may resemble measles in its characteristics, the patient does not present the eruption on the pharyngeal mucous membrane nor the peculiar coryza of that disease, nor the bronchitis or other evidences of respiratory catarrh.

The roseola of early syphilis resembles that of scarlet fever in that it first appears on the trunk; but it is not bright scarlet, but rather dusky red. It appears in patches and is not diffuse, and it ensues about six weeks or three months after the appearance of an initial lesion, occurs in an adult, as a rule, is not associated with high fever, and soon involves the face and forehead. These symptoms aid us in separating it from scarlet fever, although the rash often appears in full blast in the palms of the hands and soles of the feet; but a roseolous rash in these areas in an adult is always suspicious of specific trouble. These patches speedily change from rose-rash to other more marked lesions in cases of syphilis, and one of the first changes that they undergo is to become circinate. They fade and reappear, last an indefinite time, fade in the centre, and so change into marginate or circinate erythema.

When roseola develops after a surgical operation or after delivery in a puerperal female, it is not a manifestation of scarlet fever, but is due to sepsis, although it is, of course, possible for scarlet fever to attack such cases at any time. The rash is usually found over the abdomen and inner sides of the thighs. The absence of sore throat, the presence of a septic process, and the absence of a strawberry-tongue all help to exclude scarlatina. Sometimes late in an attack of cholera a rash like surgical roseola appears in the same areas, or in the period of reaction comes out on the forearms, backs of the hands, and rarely on the back.

The roseolous rash of typhoid is sometimes widely distributed and almost like measles in appearance; but, as a rule, it is limited to a few or many rose-spots on the abdomen, chest, or back. These rose-spots disappear on light pressure, but immediately return when the pressure is removed, and are most marked in typhoid fever about the seventh to the tenth day of the disease. They may become slightly papular. In this connection it should not be forgotten that the rose-rash of typhoid fever may be so profuse, particularly in persons with a delicate skin, as to resemble scarlet fever; and, further, it is to be borne in mind that very rarely scarlet fever and typhoid may complicate one another. The abdominal symptoms of typhoid fever and the throat symptoms of scarlet fever aid in the differential diagnosis. It should be remembered, however, that the exhaustion following an attack of scarlet fever may render the general appearance extremely like typhoid. In the relapse of typhoid fever the rose-spots often appear as early as the third or fourth day. In typhus fever they are much more plentiful and often form petechiæ.

In Bright's disease a roseola often appears over the feet and ankles, wrists, and hands, and sometimes spreads to the skin of the chest and abdomen. Desquamation may take place, but absence of febrile movements and the presence of renal trouble render the diagnosis easy. This manifestation has not a dangerous import.

A dusky-red rash rapidly spreading over the neighboring skin, above the level of which the affected area is raised, and which is separated from the sound skin by a sharp line of demarcation which can be both seen and felt, is characteristic of erysipelas. The skin soon becomes brawny to the sight and touch, and the lines of demarcation feel markedly indurated. Most commonly the disease appears on the face, starting from the inner canthus of the eye, the nostril, or the corner of the mouth. Very rarely does erysipelas affect the skin of the trunk. The fever may be quite marked, even in mild cases, and usually falls by crisis on the sixth day. In severe cases with fatal tendencies there may develop in place of crisis a typhoid state with low fever and delirium. If the disease be severe, blebs and bullæ form, the œdema of the skin becomes very profound, and finally suppuration may occur, forming what is known as phlegmonous erysipelas (see also Glanders). Erysipelatous inflammation of the skin without systemic disturbance may follow the application of arnica. A condition also closely resembling erysipelas in its raised surface is urticaria, which, however, differs so materially in other respects that a diagnosis is readily made. Aside from the absence of systemic disturbance in urticaria the swelling of the skin is not red, but pale and pearly in hue, although it may be surrounded by an erythematous blush; the onset is extraordinarily sudden, so that a skin seemingly normal at one moment, after a slight bruising by the finger or rubbing by the clothes, develops the complete eruption in a moment.

A marked roseola or dermatitis involving the insides of the thighs or the scrotum or vulva should give rise to the belief that the patient is suffering from a failure to properly pass or retain the urine, which, on escaping, irritates the skin. This is particularly apt to result if the urine is that of a diabetic. Again, it is an interesting fact that in some cases of tubercular peritonitis an erythematous rash appears on the abdominal wall around the navel.

The presence of a roseola or erythematous rash often indicates the untoward influence of some drug, following its external or internal use. We find that it very commonly follows the ingestion of copaiba, and, as many persons suffering from venereal disease take this drug, the physician must use care not to be led into a diagnosis of syphilitic roseola. It also follows the use of quinine, opium, antipyrine, and many other drugs, such as digitalis and chloral.

The roseola caused by the use of copaiba appears by preference on the upper and lower extremities, and particularly on the backs of the hands, about the knees, the ankles, and on the chest, and it is often accompanied by fever. Indeed, the eruption caused by copaiba may closely resemble a papular syphilide; but its sudden onset, itching, and disappearance when the drug is stopped separate it diagnostically from the specific disease.

The roseola following the use of bromide of potassium is, according to Veiel, very rare, and is distributed over the lower limbs. In children it may closely resemble measles.

The roseola or erythema caused by quinine is to be separated from that of scarlet fever by the absence of fever, of the scarlet tongue and sore throat, and by the fact that there are no prodromes or circulatory disturbance except the characteristic evidence of cinchonism. In doubtful cases this is still further confirmed by analysis of the urine or by the use of the following simple test. Observe the disappearance of the fluorescence of the urine caused by quinine, after the sodium chloride has been removed by precipitation by nitrate of mercury, or after separating the quinine as an iodide by the addition to the urine of a solution of two parts iodine, one part of iodide of potassium, and forty parts of water. The iodide of quinine can be again dissolved by the application of heat.

A distinct diffuse roseola sometimes follows the use of arsenic. Roseola may be caused by the use of salicylic acid and strychnine, and a scarlatiniform rash sometimes appears in blotches over the face and body in persons who are taking turpentine.

Roseola also ensues in some persons after the application of surgical dressings containing iodoform, corrosive sublimate, and carbolic acid, being due either to a local effect of these drugs or to their absorption from the dressings. Arnica tincture applied for sprains or bruises may produce marked roseola, or even erythematous and erysipelatous swelling of the skin, as already stated.

By far the most important drug-exanthem is that caused by atropine, the rash produced by it being very like that of scarlet fever, except that it lacks the red punctations of that disease. This rash may be associated with a slight rise in temperature and be followed, rarely, by desquamation. The face of a child suffering from an overdose of atropine is very characteristic. The eyes are bright, the pupils widely dilated, and the skin over the malar bones is red, but striking lines of pallor reach from the corners of the mouth to the nose. There may be active, talkative delirium and very mild convulsions from overdoses of atropine, thus making the resemblance to the onset of scarlet fever very striking. The brief duration of the rash, its lack of punctation, the absence of high fever, and the history of the patient having taken atropine or belladonna, all help to make the differential diagnosis.

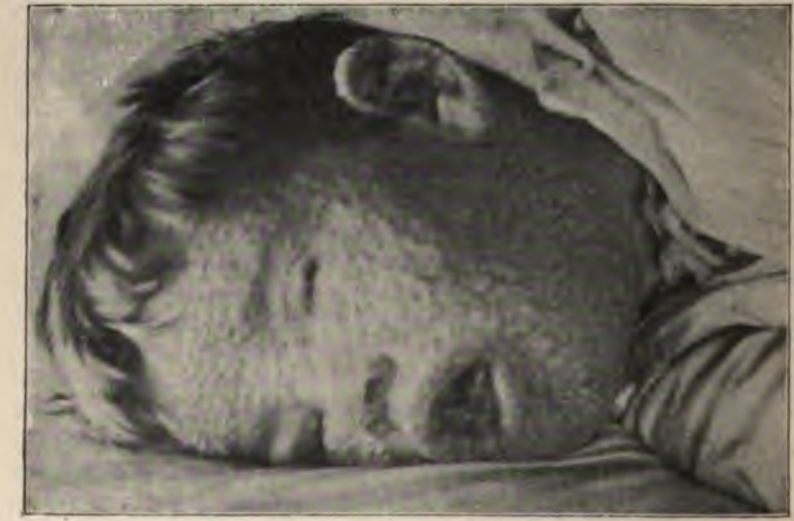
Roseola, followed by desquamation, has been known to follow the hypodermic injection of mercury. Sometimes the use of blue ointment produces a widespread rash resembling measles, and this resemblance may be increased by the development of a febrile movement. A similar eruption may ensue from the ingestion of opium.

Erythematous rashes, too, frequently follow slight irritation of the skin in persons who use chloral.

Acne of the skin, particularly on the face, is often produced by the use of bromide or iodide of potassium, or of any preparation containing bromine or iodine. That produced by iodine is generally sudden in its onset and profuse in its distribution. The base of the pimple is bright red, the top speedily becomes pustular, and Fournier states that it may be hemorrhagic. Stopping the ingestion of the drug speedily relieves, or at least decreases, the eruption. The acne due to bromine is often very profuse, and the pimples in severe cases may coalesce, making sloughs of considerable size with an indurated base.

In some persons, generally females, there is developed an acne on the face, breast, and back, as the result of taking iron as a tonic.

FIG. 68.

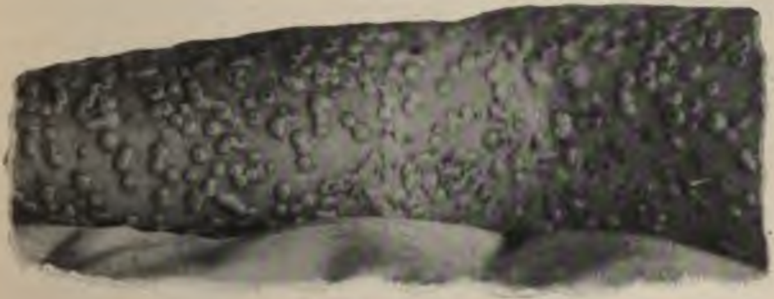


Smallpox eruption on the seventh day.

In addition to the acne caused by drugs or their compounds, we should also mention the acne and furuncles appearing in persons working in paraffin, which is due to blocking of the sebaceous glands.

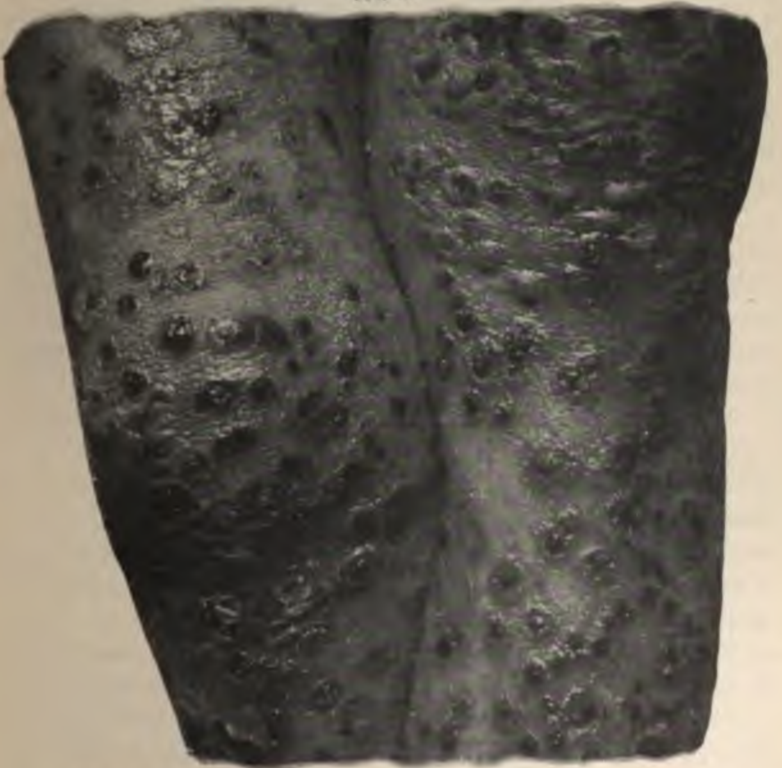
Smallpox. Closely associated with this form of eruption is that which is characteristic of smallpox and chickenpox. The eruption of smallpox appears on the second or third day in the form of tiny specks, resembling flea-bites. These rapidly become papules, which have an indurated base, so that they feel as if shot were under the skin. (Fig. 68.) After about thirty-six hours these papules become vesicles, containing a turbid fluid, which speedily becomes purulent, forming a pustule. (Figs. 69 and 70.) Generally this process of

FIG. 69.



Smallpox eruption on the eighth day.

FIG. 70.



Smallpox eruption on the eleventh day. The pock is seen to be umbilicated.

maturation takes three days, and, with the development of the pus, the so-called secondary fever, which may be even higher than the primary fever of invasion, sets in. After a period of eighteen to twenty-one days the pustules drop off, having become dried up, leaving, if the attack has been severe or the skin delicate, deeply pitted scars. The vesicles of variola soon become umbilicated, are multilocular, and are difficult to rupture with the finger-nail. Although the eruption of smallpox appears on the forehead, which is the favorite seat of acne in many cases, a differential diagnosis is not difficult, since the grave systemic disturbance, febrile movement, and rapid involvement of the skin of the limbs speedily indicate the true nature of the disease. The early appearance of the rash on the hands in variola is a valuable diagnostic sign, as acne in this part of the skin is practically unknown. Then the sudden development of the eruption in smallpox is entirely different from the gradual onset even of the most intense acne.

It is not to be forgotten that cases of smallpox develop in which the symptoms of systemic disturbance are so mild that it seems impossible for true smallpox to be present, and also that acuminate syphilide accompanied by febrile movement may develop about six or seven months after infection, which may be accompanied by such marked systemic disturbance as to resemble smallpox.

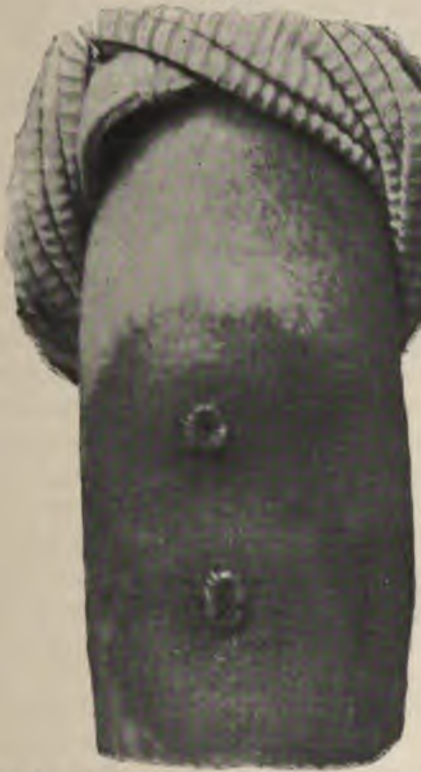
Pustular syphiloderm sometimes resembles smallpox so closely as to almost defy an immediate differentiation. The history of syphilitic infection or of certain syphilitic eruptions, and the absence of severe systemic symptoms, may aid us, but fever may be present. As the pock develops in syphilis it does not become so well umbilicated, nor does it leave deep pits in the skin.

In some cases a purulent acne of the forehead develops in syphilis.

The separation of variola from measles has already been discussed, and it is only in the papular stage that the former disease can be confused with the latter, while the reddened mucous membranes and swollen face of the case of measles soon determine the diagnosis. The rapid formation of vesicles and the shot-like sensation of the eruption show that the rash is not measles.

Vaccinia. The appearance of the eruption of vaccinia following vaccination must be next described. Three or four days after the vaccination a single or several papules arise on the scarified surface, which by the sixth day are changed into umbilicated vesicles, which soon unite and form one vesicle the size of a five-cent piece. This vesicle finally forms a scab, which falls off after the expiration of about three weeks from the inoculation. A "good primary take" is always surrounded by an areola of rosy red of several inches in width. Rarely severe inflammation and sloughing ensue. (Fig. 71.)

FIG. 71.



Typical vaccine vesicles of a "primary take." Tenth day.

When the vaccination is a secondary one, the "take," if it occurs, often produces no symptoms until the ninth and tenth day, and the local lesions are then very mild.

Chickenpox. In chickenpox the eruption appears on the first or second day, and keeps coming out for several days. It is rose-colored and occurs as papules, which immediately become vesicles. They last but four or five days, which is the time that it takes the eruption of smallpox to develop, and are usually associated with very mild febrile disturbance, the child remaining but little indisposed if well cared for and nursed. Unlike smallpox, varicella does not become umbilicated unless it grows about a hair follicle which holds the centre of the pock, and rarely leaves pits in the skin unless the vesicles are picked at by the finger-nails. Neither do the vesicles become pustules unless infected by picking or the child is in a condition of debility or suffers from struma. Varicella is separated from variola by the absence of severe systemic disturbance, by the rash first appearing on the chest and neck instead of the forehead and hands, by the presence of other cases of the disease in an epidemic, by the repeated crops of the eruption in vaccinia, so that several sets of vesicles may be present at one time, and, finally, by the fact that it attacks children who have been well vaccinated, whereas smallpox does not. The history of exposure is, of course, an important point to be investigated.

The profusion of the eruption is not of diagnostic aid, as it may be scant in variola and profuse in varicella.

An eruption somewhat resembling chickenpox or smallpox is that called impetigo contagiosa, in which there are found multiple, flattened or slightly umbilicated, roundish or oval vesicles, pustules, or blebs, which form after some days dry yellowish crusts. It occurs in childhood or early adult life, and is often associated with some degree of fever. The areas involved are the face, neck, buttocks, hands, and feet. The lesions of the skin are larger than in chickenpox, but often follow this disease. As its name indicates, the disease is contagious, and the occurrence of a series of cases in close proximity to one another should not mislead the physician into a diagnosis of variola or varicella. The eruption lasts about two weeks, and Kaposi asserts that swelling of the submaxillary glands is always present. We can further separate impetigo contagiosa from varicella by the localization of its eruption to one area, as a rule, by the fact that the eruption becomes bullous or purulent, and by the larger size of the vesicle. From smallpox we can separate it by the absence of severe pain in the back, the grave systemic disturbance, and the secondary fever of that disease, accompanied as they are by the smallness of the pox, the peculiar odor of the patient, and the history of exposure to variola.

In the presence of a papular, pustular, or vesicular eruption of the skin it must be remembered that quinine sometimes develops these lesions in susceptible persons. In some instances where it involves the hands it may indicate that a local effect has been produced by working with the drug.

Eczema in its various forms may appear as the result of the use of quinine internally or locally, or of the employment of mercury internally or externally. When it arises from the use of iodide of potassium, which is very rare, it chiefly affects the scalp and scrotum. The development of an eczematous irritation of the skin sometimes follows the use of chloral.

Herpes labialis is a very constant lesion associated with croupous pneumonia, and its development is said to be a favorable sign. It is also an important sign for the separation of epidemic cerebrospinal meningitis from meningitis due to other causes, as it is not commonly present in the non-epidemic form. It sometimes arises as a result of using salicylic acid.

The presence of herpes rather excludes tuberculosis, typhoid fever, and simple pleurisy from the case in instances in which the diagnosis is doubtful, since it is rarely met with in these maladies.

The development of recurring crops of boils in persons not exposed to paraffin or tar should cause the physician to suspect the presence of diabetes mellitus, or at least that there is general debility, and particularly an absence of lime salts from the system in the proper quantity. When the ordinary boil passes into a condition of marked induration about its base, with sloughing of the subcutaneous tissue and necrosis of the skin, which becomes perforated by the openings of several sinuses, we have to deal with a carbuncle or anthrax simplex. The disease usually appears on the back of the neck, on the back, or the lip. The systemic disturbance is very great and the exhaustion profound. The skin covering the area involved becomes grayish or bluish-black, and then separates as a large mass, while the subcutaneous tissue comes away in shreds. It is a dangerous disease in all persons, but particularly so in those who are already weakened by other diseases or excess.

The development of a painless macule on the skin of the hand or foot, followed by an acutely inflamed papule which itches and is soon changed into a relaxed vesicle containing bloody serum, in which there is a hard nucleus which rests upon an indurated base, is the initial manifestation of anthrax maligna or malignant pustule. The lymphatics soon become swollen, and metastatic abscesses speedily form elsewhere, as in the axillary glands. The systemic symptoms are severe, sometimes being manifested in high fever, in other cases by a typhoid state. Death is very commonly the sequel (65 per cent.), even if prompt surgical interference takes place.

There is generally a history of exposure to infected animals or their hides. Malignant pustule is to be separated from carbuncle by its fulminating character and peculiar appearance.

When an erysipelatoid rash with swelling of the skin and the development of papules, vesicles, pustules, and bullæ appears in association with induration of the skin, with sloughing eventually taking place, the disease may possibly not be erysipelas of a phlegmonous form, but glanders or equina. Numerous inflammatory foci appear in the skin in glanders which end in local abscesses and hemorrhagic nodules, and profound systemic infection is always present. The presence of a sanious discharge from the nose aids in confirming the diagnosis. Death usually comes in a few days in this acute form. Should the course of glanders be chronic, pustules somewhat like those of smallpox, except that they are not umbilicated, lie on an indurated base, and in them is formed a viscid or sanious pus of offensive odor. This disease is rare. Both forms arise from infection from a horse suffering from the malady. Glanders may be confused with variola or the pustular and ulcerative gummatous stages of syphilis.

The development of pea-sized or larger bullæ upon the skin may indicate the presence of pemphigus, or if there is central nervous disease involving the spinal cord and resulting in trophic lesions similar bullous eruptions may take place. The bullæ, if they contain dark bloody fluid and are situated upon a limb in which there is an abnormally high temperature, are peculiarly indicative of central nervous lesions, particularly if there is a tendency to dilatation of the capillaries of the skin on slight irritation; but if the temperature of the entire body be raised, the physician should remember that pemphigus is a disease in which there is often marked febrile movement. Sometimes these bullous manifestations are followed by gangrene in cases of neuritis or other disease causing trophic lesions, such as myelitis and paretic dementia.

Bulæ on the face may follow the ingestion of antipyrine or iodine compounds.

The development of a pemphigus-like eruption in the skin may follow the use of salicylic acid or copaiba.

In the cases of herpes zoster the skin lesion often has its origin in compression of the spinal cord, or in such diseases as tabes, spinal meningeal irritation, and peripheral neuritis.

Glossiness of the skin, in which its minute creases become smoothed out and it appears unduly shiny, often results from chronic disease involving some portion of the nervous system connected with the government of nutrition. Very commonly it results from peripheral neuritis. In addition to glossiness there are often redness and marked thinning or thickening of the cuticle and subcutaneous tissues.

Gangrene of the skin may follow nerve injuries or central nervous lesions. Thus, it may follow upon division of a nerve-trunk, or be due to cerebral abscess, in which case the gangrene will be with the other localizing symptoms on the opposite side of the body.

The cerebral form develops suddenly and without the prodromal redness of bed-sores as seen in prolonged illnesses. Similar rapidly developing sloughs and ulcerations of the skin are seen in cases of acute myelitis and in the second and third stages of paretic dementia.

A very interesting condition is the so-called spontaneous gangrene of hysteria. On the skin, generally of the breast of a young girl, a spot develops which feels to her to be hot and burning. The skin soon becomes very white, then in a few hours very red and forms a wheal. This rapidly becomes dark and bluish-black, looking like a burn of sulphuric acid, and a slough finally comes away, leaving a permanent cicatrix.

Gangrene of the skin follows upon diabetes mellitus, and may involve the scrotum or vulva if the irritation of these parts by the urine is constant. More commonly the toes are affected, and there is this important differential point, that in the gangrene of old age with bad vessels the lesion is usually at the tip of the toe, whereas in diabetic gangrene it is frequently about the ball of the big toe or on the sole or dorsum of the foot. Previous to the development of gangrene there are developed bullæ and other inflammatory changes in the skin which is about to be affected. Kaposi describes a serpiginous form of gangrene affecting the leg in diabetics and a variety of tissue break-down in which a dermatitis, followed by ulcers and a lupus-like formation, also occurs in diabetes. Perforating ulcer of the foot occurs in locomotor ataxia and in paretic dementia.

Closely related, yet quite distinct from angioneurotic œdema, is that condition called Raynaud's disease, symmetrical gangrene, or local asphyxia, according to its severity. The fingers and toes or the nose, with or without exposure to cold, are found to be pale and livid, looking like a hand from which all the blood has been removed by the use of an Esmarch bandage. The part often feels as if "asleep," and is more or less numb and without sensation. To the touch the part is cold and waxy, and it does not bleed when pricked. With the onset of these signs there are often general chilliness and malaise. Often this manifestation speedily disappears, leaving the skin apparently normal; but if it persists, the skin becomes glossy, shrivelled, and looks as if it had been soaked in hot water for hours. When the disease is more severe the pale waxiness is supplanted by cyanosis till the finger-tips, for example, look as if dipped in blue ink; there is often local swelling; the skin is frequently found to be sweating freely and is distended with blood. The skin may rapidly separate from the deeper tissues and become necrotic in patches or *en masse*, and the entire tip of the finger, after becoming black, shrivels up into a condition resembling dry gangrene, which is separated from the sound skin by a sharp line of demarcation. Sometimes small necrotic patches slough out, which leave cicatrices telling of the attack. The prognosis is not bad. The most interesting complication of the disease is paroxysmal hæmoglobinuria.

The development of gangrene of the fingers and toes sometimes follows the prolonged use of bread made from rye which is infected by ergot.

Sometimes gangrene of the skin follows severe attacks of the exanthemata in children who are strumous or very feeble, or who are syphilitic.

Ulcers about the base of the finger-nails should rouse the suspicion of the excessive use of chloral.

Bed-sores may develop whenever by long-continued pressure upon any part of the body the local circulation is disturbed, particularly if in addition there is general systemic debility from some exhausting disease, such as typhoid fever. They also develop very speedily, and apparently almost spontaneously, in the course of acute transverse myelitis. Under these circumstances the sacrum is the area most severely affected. Sometimes these sloughs have been known to develop as early as six hours after the beginning of the attack. Associated with the involvement of the soft tissues the bones may break down, and cellulitis about the rectum and bladder place the patient's life in immediate jeopardy. In hemiplegia, particularly in that which is due to cerebellar hemorrhage, bed-sores often form on the buttocks, and in paraplegia from other causes than transverse myelitis, upon the sacrum. They also appear on the heels, inside of the knees, and about the hips in some cases of paraplegia.

Sudden sloughing of the skin of the nates sometimes occurs in cases of intracranial hemorrhage, and is said by Joffroy to be connected with lesion of the occipital lobes.

The value of roseola and rupial eruptions in the diagnosis of syphilis has already been dwelt upon. When the roseola becomes transformed into slightly elevated or bean-shaped spots, irregularly scattered, but sometimes forming groups which are apt to be circular, and these circles become marginated and then scaly on the edges, resembling lepra or psoriasis, or even go further than this and develop bullæ and blebs, and when the sores which form are filled with a clear liquid which may become sanious or turbid and on drying leave crusts, the removal of which reveals deeply excavated sloughs, the area of the slough often being as large as a silver dollar, but often irregular in outline, syphilitic rupia is probably the lesion. There is, however, this important differential point, namely, that in specific rupia there is an essential feature, a peripheral ring of induration, whereas in the non-specific form this induration is absent.

If, in addition to these variations, the eruptions are dusky red and leave behind them on healing copperish-looking discoloration of the skin, and appear on areas, such as the flexor surfaces, where ordinary skin eruptions are rarely seen, the diagnosis of syphilis is highly probable. If the eruption is chiefly tuberculated and the tubercles are large and more marked than usual, and if they ulcerate and become deep sores, and finally form on healing well-marked cicatrices, tertiary syphilis is to be considered the probable cause.

If, again, we find small nodules under dusky-red skin, which finally breaks down and discharges bloody serum, or pus which in burrowing forms discharging sinuses, syphilis of the third stage may be regarded as a likely cause.

The appearance of hard, dark-brown, infiltrated areas in the skin may be due to the excessive use of bromine, and as they gradually become depressed in the centre closely resemble in some cases the nodules of syphilis.

The skin of the abdominal walls in cases of ascites is apt to be not only thinner than normal, but tense and slightly shiny, while its appearance when viewed in a good light may be slightly blue like the iridescence of certain kinds of glassware.

Scars of the skin often give us much useful information.

Early tendencies to struma or tuberculosis may be found in the scars resulting from suppurating cervical glands. In the groin such scars may be an evidence of venereal infection, although it should be remembered that suppuration of these glands usually takes place as a result of chancroids and not from true chancre. It has already been shown that syphilitic skin lesions often leave scars to mark their site. Scars upon the head tell us of possible injuries to the brain in suspected traumatic epilepsy, or of falls in epileptics. Similarly, other traumatisms in the history of the patient may be discovered by scars elsewhere.

The presence of numerous regularly arranged fine scars on the chest or elsewhere may develop the fact that the patient has at some time been wet-cupped for some pulmonary or other disease; or if the peculiar three-pointed scar of the leech is seen, another good evidence of a bleeding is presented.

When the skin of the abdominal wall exhibits striæ or scars arranged in parallel series, it indicates that it has been stretched very considerably by pregnancy, ascites, or, more rarely, by excessive corpulence. Sometimes these striæ appear on the lower limbs in pregnant women or in persons with dropsy. Very rarely they may develop on the arms or legs or elsewhere during convalescence from some grave disease, such as typhoid fever.

Sweating of the skin, aside from the normal and almost imperceptible exhalation of moisture, takes place in health as a result of severe muscular exertion, whereby the peripheral circulation is increased and the bodily temperature raised, or when the body is very heavily clad or exposed to external heat in excess. In all these cases the sweating is to be regarded as a physiological effort on the part of the body to reduce its temperature by increased evaporation from the surface. In disease sweating provides us with very important information in many conditions.

During the course of fevers which naturally end by crisis the occurrence of a profuse sweat (generally associated with a fall of temperature) gives us the first sign of beginning convalescence, and in irritative fevers, or those due to cold and congestion, the artificial production of sweat is decidedly a good omen. The sweat of crisis is perhaps most marked in croupous pneumonia. Profuse sweating is also a characteristic symptom of relapsing fever, pyæmia, acute ulcerative endocarditis, phthisis, malarial fever of the distinctly periodic type, and of typhoid fever and collapse. Constant profuse sweating is marked in some cases of acute articular rheumatism, and it is worthy of note that, while sweating generally occurs in febrile diseases at a time when the temperature is falling, in rheumatism the febrile movement may even increase during the sweat rather decrease.

Profuse so-called colliquative sweats often occur at night in debilitated persons without the presence of any febrile movement, and are an evidence of profound nervous and vasomotor relaxation. Moderate sweating sometimes is seen from similar causes in feeble persons after taking anything in the food or drink which produces circulatory or nervous excitement. Localized sweatings occur almost solely in subjects of nervous disorder, which is often organic, as in paretic dementia, and sometimes functional, as in hysteria or Raynaud's disease. They depend upon perverted vasomotor influences sent to the glands and their supplying vessels in particular areas. Localized sweating of one side of the face or neck or chest is often a most important sign of a thoracic aneurism pressing on the cervical sympathetic. Bromidrosis may occur in hysteria, or the head may be the only part affected in Graves's disease and in migraine. Profuse sweating of the head of an infant when sleeping may be indicative of rickets.

In cases of the uræmia of cholera or of renal disease there may be profuse sweating, which takes the place of the dry and hot skin seen more commonly in this condition. The surface of the entire body is usually involved in the sweat.

The quality of the sweat varies greatly in many persons. In cases of deficient renal activity it often contains urinary elements, smells uriniferous, and may even deposit particles on the skin in small white scales, particularly on the forehead and nose. This is called uridrosis. In jaundice the sweat may be bile-stained.

Excessive dryness of the skin is seen in grave forms of renal disease, in nearly all acute fevers with a high temperature, and in cholera and diabetes, in which diseases the dryness is largely the result of drainage of liquids from the body.

Sometimes after a prolonged dryness of the skin during high fever, as soon as sweating begins hundreds of little blisters develop, due to retained sweat under the epiderm. These are called miliara or sudamina.

When the skin is dry and harsh, and the naturally thickened portions have in their folds a peculiar white appearance as if filled with meal, diabetes should be sought for. Rarely the physician may be deceived by profuse sweating in diabetes, in which disease the skin is usually very dry.

Dropsy and Swelling of the Skin. Swelling of the skin and subcutaneous tissues occurs most frequently as a result of dropsy, in which condition the lymph-spaces become filled by liquid. The skin in the area involved is not only swollen but doughy, or if the effusion is very great the skin may be of almost board-like hardness, so tensely is it distended. Pressure with the tip of the finger upon such an area will result in pitting, and this is one of the more important signs separating dropsy or true œdema from the swelling of acute inflammation, which, while it may be very tense, does not pit. Further, the swelling of inflammation is usually localized, reddened, and feels hot to the touch, whereas the dropsical swelling is more diffuse, is pale, and the temperature of the part is lower than normal.

When the effusion of liquid is limited to one portion of the body it is usually called œdema or localized dropsy, whereas if the entire body is boggy it is designated general anasarca. Dropsy is to be differentiated from myxœdema by the facts that in the latter disease the onset is very slow, the swelling does not pit on pressure and is universal and fairly equally distributed over the body, the thyroid gland will often be found diseased, the subcutaneous tissues are not boggy but resistant, and there is anæsthesia of the skin. When the subcutaneous tissues are distended by air, instead of liquid, they are even less resistant than in dropsy, the swelling is usually very localized and does not pit, and the part crackles or crepitates on gentle pressure.

The presence of dropsy is indicative of many widely separated diseases. In the first place, it may indicate a deficient circulation of blood, either by reason of a feeble or diseased heart or because of obstruction by the pressure of growths, thrombi, or emboli.

It may be due to disease of the walls of bloodvessels and lymphatics, as is generally the case in renal disease, or it may arise from disease of the blood itself. Again, in some cases it is due to disordered nervous control of the vessels, by reason of centric or peripheral changes which may be organic or functional.

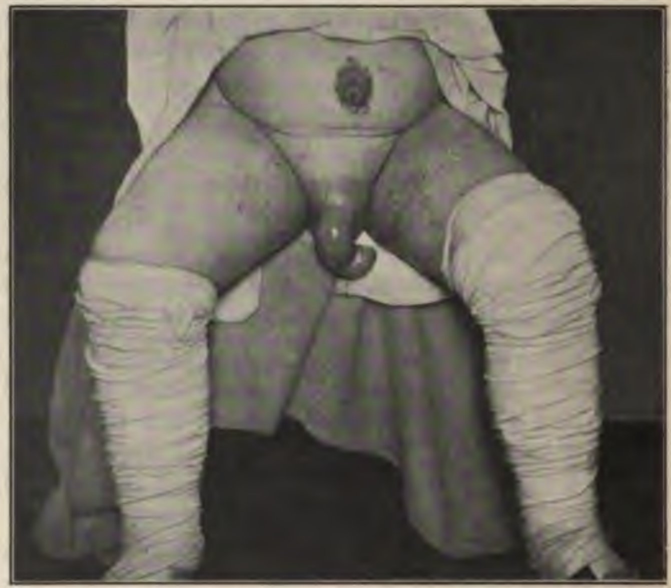
The significance of a widely diffused general dropsy or anasarca is generally that there is well-marked renal disease, and this probability is greatly strengthened if the œdema of the face be well marked, particularly in the morning on arising, disappearing as the day goes on. The skin in such cases will usually be quite pale, and an examination of the urine will reveal the presence of the signs of nephritis. The next most common cause of general anasarca after renal disease is heart disease. When due to this cause it will be found that the ghastly pallor of renal anasarca is replaced by cyanosis, and often by engorgement of some of the superficial veins, while the physical signs of cardiac disease will confirm the diagnosis. General anasarca may rarely arise as a result of a multiple peripheral neuritis, and it also occurs as a symptom of beri-beri and from the excessive use of large amounts of arsenic. This arsenical anasarca may be due to the neuritis produced by the drug, although Wood thinks it is due to a cellulitis. Rarely we find general anasarca in cases of advanced cancerous cachexia, and care must be exercised that the hæmic murmur due to anæmia does not mislead the physician into a diagnosis of heart disease.

Dropsy widely diffused or localized in the feet and legs also occurs in scurvy.

The most common seat of localized dropsy or œdema is the feet and legs, particularly about the instep, the ankles, and the tibiæ. When it is bilateral it is generally indicative of cardiac failure or more rarely of renal disease. Nearly always, if it be renal, a careful examination will discover œdema in other parts of the body, although it may be most marked in the feet and legs. In many cases the various serous sacs, such as the pericardium, peritoneum, and pleuræ, will be found to contain more liquid than normal, and the tissues generally will be found infiltrated.

Other causes of œdema of the feet and legs are anæmia, and obstruction to the return of blood from the lower limbs by reason of growths in the abdomen pressing upon the iliac veins or inferior vena cava. Thus, cancer of the pancreas sometimes causes œdema of the feet and legs in this manner. Very rarely œdema of the lower extremities follows hepatitis or hypertrophic cirrhosis of the liver as a primary symptom. Usually such lesions produce ascites alone, or if the legs are involved they become so by reason of the pressure of fluid in the pelvis during the time that the patient is sitting up or standing. This latter cause of bilateral œdema of the lower limbs is, however, rare. Sometimes œdema of both legs and feet comes on in persons who, though feeble and relaxed, remain standing with little muscular movement during many hours in the pursuit of their occupation, as in type-setters and salesmen, or in young persons who have subjected themselves to excessively severe muscular exercise. In other instances, very much more frequently, œdema of the feet and legs comes on in the course of profound anæmia resulting from slow hemorrhages or other causes. It is also seen in the cachectic stage of cancer, owing to the anæmia which is present. General swelling of a leg in a puerperal woman is probably due to phlegmasia alba dolens, and this affection may also be bilateral. Both Herman and Cameron Kidd have each reported a case of bilateral phlegmasia alba dolens occurring in a virgin with anæmia. When it occurs in males it is most commonly unilateral and a complication of convalescence in typhoid fever. It is due to thrombosis of the left femoral vein, as a rule.

FIG. 72.



Intense œdema of legs, scrotum, penis, and abdomen due to mitral stenosis. The distortion of the penis due to the effusion is characteristic.

When the face is œdematous the swelling is most marked under the eyes, the lower lids of which are particularly puffy in the morning and nearly normal in appearance at night. This form of œdema is most marked in, and is almost pathognomonic of, renal disease. Its only other causes are the excessive taking of arsenic and angioneurotic œdema. More alarm should be felt at a slight swelling of the face of this character than if the feet are markedly puffed. Sometimes œdematous swelling of the side of the face and scalp which has been involved in a severe attack of neuralgia takes place.

When œdema of one or both eyelids occurs, with protrusion of the eyeball, the swelling extending to the rest of the face as time goes on, it forms an important symptom in obscure cases of suspected cerebral thrombosis, and is caused by the intimate association between the intracranial vessels and those of the face.

Sometimes œdema of the eyelids comes on in neurotic subjects and may extend to the forehead. This may be seen in children, most commonly about puberty, and is probably the result of a neurosis.

Œdema of the upper extremities alone only results from causes interfering with the flow of blood, such as are produced by morbid growths in the chest, as mediastinal growths, and in cases of aneurism. When the swelling is limited to one arm or leg it is a sign that there is interference with local circulation, as, for example, the obstruction of the femoral vein by thrombus, as in phlegmasia alba dolens following labor or enteric fever, or, when the œdema is in the left leg, by cancer of the sigmoid flexure. If the swelling of the arms and head is manifested suddenly, it may be due to that rare condition in which an aortic aneurism ruptures into the vena cava; whereas if it develops slowly, it is due to pressure by a growth.

There remain three forms of local œdema of some diagnostic significance, namely, that occurring in a limited area over some deep-seated suppurative process, as in the skin back of the ear in cases of mastoid abscess or thrombosis of the lateral sinuses, that over the ribs in cases of purulent exudation into the pleura, and that on the thigh in the deep muscular abscesses which sometimes follow typhoid fever.

Œdema of the legs and wrists sometimes complicates relapsing fever, and is evidence of profound feebleness, without necessarily indicating renal or cardiac disease. Such a limited œdema, or even general anasarca, may occur during convalescence from typhoid fever from similar causes. Unless this effusion is associated with signs of grave renal or cardiac mischief the prognosis, according to Lendel, is favorable.

No better place can be found in which to mention that condition in which irregular and numerous masses of fat are to be found in the subcutaneous tissues of middle-aged persons, usually women; these masses are more or less painful, and are found in the limbs and extremities. The skin itself is not altered. Dercum first described this state and gave it the name of *Adiposis dolorosa*.

When the skin is pale and affected by an œdematoid swelling, with thickening, hardening, and loss of elasticity, particularly about the face, and also in the trunk and extremities, and if this swelling, which resembles œdema, fails to pit on pressure, the physician should remember that myxœdema or the cretinoid œdema of Gull may be present. If in addition to these signs there is a half-idiotic or heavy expression of the face, a slow and labored manner of speech, with thickened, clumsy fingers, the diagnosis is made practically certain. The brain in this disease perceives or grasps ideas very slowly, and all the functions of the body seem torpid.

There are several other diseases in which great thickening of the skin takes place, which cannot, however, be confounded with myxœdema. In elephantiasis there is a hypertrophy of skin and subcutaneous tissues which is confined to some particular region of the body and arises from local circulatory disturbance in the blood and lymph-vessels. The skin is very hard, so that the leg, if affected, feels like a solid mass of wood. The disease most commonly affects one of the legs, rarely both, and the scrotum. In both myxœdema and elephantiasis the process develops very slowly.

When the skin is dotted with irregular patches or streaks, which may be depressed, elevated, or tightly stretched, or if the entire skin is thickened, covered with thin scales, or possesses a plaster-like appearance, the physician should recognize these symptoms as indicative of scleroderma. If in addition to these signs there is a fleeting pitting of the skin on pressure, and it cannot be pinched into a fold, the diagnosis is confirmed. Sometimes the skin in scleroderma seems bound down by tense cords or bands of retracted connective tissue to the tissues beneath, and in rare instances even the tendons, muscles, fasciæ, and joints may be involved. Generally the impaired movements of the joints depend upon the stiffening of the skin, but in some cases the disease results in atrophy of these deeper parts.

If during the first months of life the skin of an infant becomes œdematous, hard, tense, and glossy, varying in color from a white to a reddish or dirty yellowish-brown, and if this rapidly involves the entire surface so that the integument becomes cool, immovable, and resistant, the child appearing as if frozen into stiffness, it is probably suffering from sclerema neonatorum, a disease entirely different from the scleroderma of the adult. As a rule, death speedily ensues, but before this takes place the parts first affected become thin and lose their swelling and may develop cyanosis and gangrene.

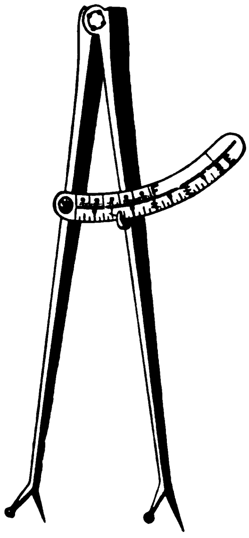
The affection just described is to be separated from œdema neonatorum, a condition arising in prematurely born children. Within a few days after birth there is discovered a pallid, cold condition of the buttocks, thighs, legs, and arms. The parts speedily become œdematous and livid blue. Finally the œdema may become very marked and the skin tense in consequence. Intense drowisness is a characteristic of the disease. Death commonly ensues, but recovery may occur. While the color of the skin may be identical in œdema neonatorum and sclerema neonatorum, the former affection lacks the stiffness of the jaw and other joints, and the pitting on pressure is marked. As scleroderma does not occur before the first year, it can be excluded from the diagnosis.

Very closely allied in its causes and appearance with urticaria of the severe type is angioneurotic œdema. In this condition there appear upon the skin numerous patches or plaques of circumscribed puffy swellings, which have a red appearance and vary from the size of a nickel to a silver dollar or larger. There is an absence of itching, an important difference from true urticaria, but the part affected may be tense or hot to the patient. These attacks are generally recurrent, and take place in neurotic persons. They may cause loss of sight through swelling of the eyelids and, where the mucous membranes of the pharynx and larynx are involved, serious interference with breathing. The swelling of angioneurotic œdema does not pit, and it is to be separated from the blue œdema and white œdema of hysteria. True angioneurotic œdema is rare in hysteria, and if localized swellings do result from this condition the physician will generally find marked hysterical signs manifested, such as disorders of sensation or tenderness over the ovaries.

The ocular appearance and touch of the skin having been studied in so far as its surface affords evidence of more deeply seated disease or functional disturbance, we next pass to a study of its sensibility, having the same diagnostic objects in view.

Sensation in the Skin. Before considering the various perversions of its sense it is important to remember that the sensibility of the skin may be divided into four parts, namely, its tactile sense, its pain sense, its thermic sense, and its sense of pressure. Any one of these senses may be perverted or in abeyance without the others being affected in a similar manner, and it is noteworthy that, while corresponding areas of the skin in all individuals have practically identical sensibilities, each part of the skin has a sensitiveness of its own, so that while in some parts the slightest touch is felt, in others severe irritation must be produced to cause much of a result. These differences have been carefully studied by many observers, the most thorough being Weber, who has found that the average ability to separate points brought in contact with the skin is about as follows: at the finger-tips points can be separated at from 2 to 3 millimetres, on the lips 4 to 5 millimetres, on the tip of the nose 6 millimetres, on the cheeks and backs of fingers 12 millimetres, and on the forehead 22 millimetres. The skin on the neck separates points at 34 millimetres; that on the forearm, on the lower leg and back of foot, at 40 millimetres; on the chest at 45 millimetres; on the back at 60 millimetres, and on the arm and thigh at 75 millimetres. If tests be frequently repeated in a single individual, the ability to separate the points increases with training. Care should always be taken that the pressure on both points is equal, applied simultaneously, and that the points are equally sharp.

FIG. 73.



Carroll's æthesiometer.

In testing tactile sensibility, not only should points be used, but also objects. Often single points may be applied without any abnormal manifestation, and, in some cases of disease, the skin, which seems devoid of sense on ordinary touch, is found to be excessively hyperæsthetic if the hand is drawn lightly over it.

The best apparatus for testing tactile sensibility is the æsthesiometer of Carroll, which is a pair of double-pointed compasses connected by a graduated scale. (Fig. 73.)

The ability to distinguish pain-giving and thermal applications is most acute in the normal skin of the hands, in which tactile sense is also most acute.

The methods by which we test the pain sense are several, but chiefly by pricking the skin, more or less deeply, with some sharp-pointed instrument, such as a pin, or by pinching the integument.

The thermal sense is studied by appplying bodies which are hot or cold against the skin, such as a cold knife, a small piece of ice, or a test-tube which contains very cold or hot water. In all such tests the physician should use both hands simultaneously. With one hand he should apply his instrument to the suspected area, and with the other a similar instrument to an area known to be healthy, in order that an actual comparison as to the sensations may be noted by the patient. Thus the face may be used as the normal area in a spinal lesion, and the skin of the arms as a control-surface in a lesion involving the legs. The eyes of the patient should be blindfolded, and if tactile sense is being tested the instrument must be of the same temperature as the body.

FIG. 74.

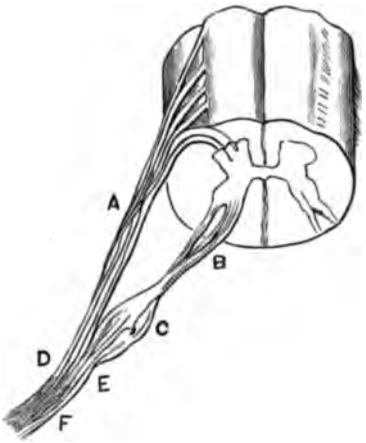


Diagram of the relations of the nerve-roots to the spinal cord. A, anterior root; B, posterior root; D, motor part; E, sensory part; F, mixed nerve. (EDINGER.)

Closely connected with the subject of tactile sense is what is known as stereognosis, or the ability to recognize objects by contact and grasp. By this means healthy persons are able without looking at an object to judge of its character, and in the blind this sense is very highly developed. Manifestly the entire sensory apparatus must be intact for stereognosis to be performed, since any interference with the sensory system will produce that condition which is known as "astereognosis." Not only does stereognosis involve the tactile sense but also muscle sense, since it is by the grasping of the object that information concerning it is gained, as well as by its coming directly in contact with the hand. At the present time the discovery of astereognosis in a patient has not very definite clinical significance, so far as localization of the lesion producing this condition is concerned. When astereognosis does not depend upon a disease of the sensory nerves, it is commonly supposed to depend upon involvement of the cerebral cortex, in the parietal lobe, if the sense of weight, or of the position of the fingers, or if ataxia of movement without signs of peripheral nerve or cord disease is present. Of course it is possible for a lesion in the sensory fibres of the internal capsule to cause astereognosis, but, on the other

FIG. 75.

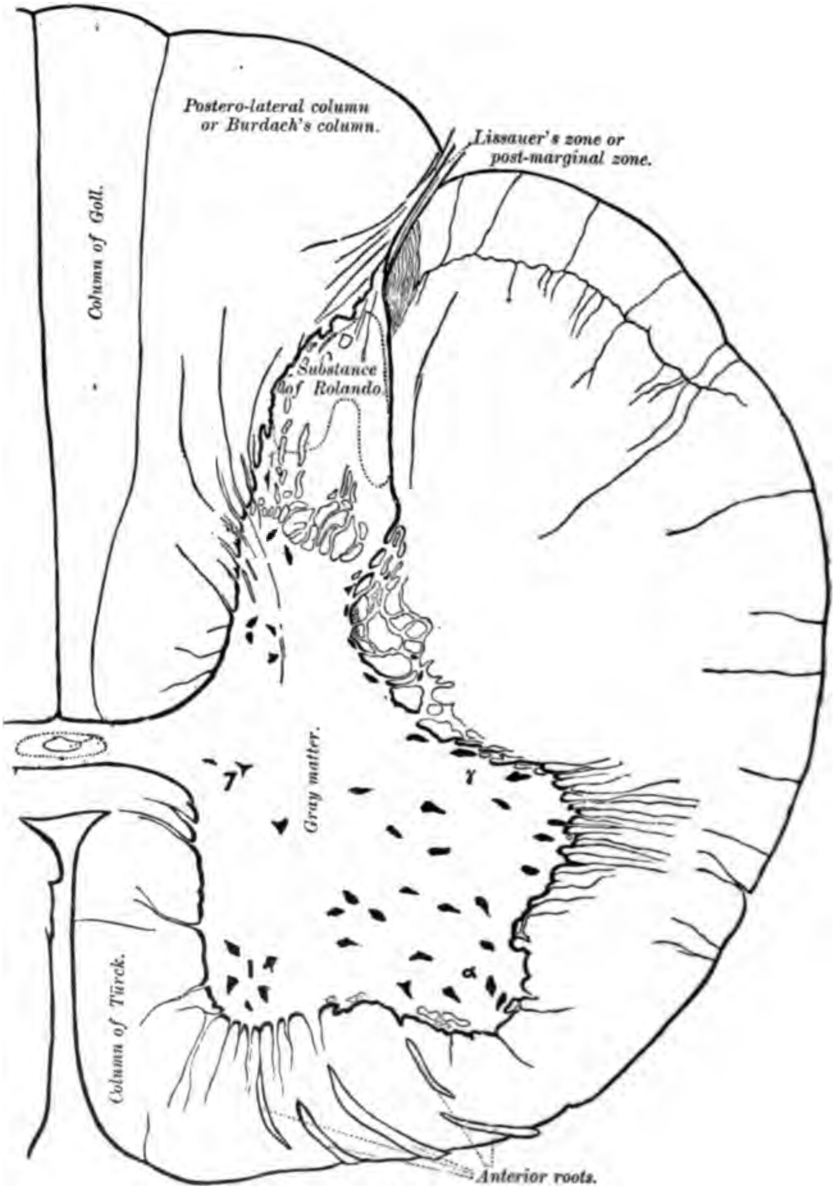

Postero-lateral column or Burdach's column.
Lissauer's zone or post-marginal zone.
Column of Goll.
Substance of Rolando
Gray matter.
γ
γ
α
Column of Türck.
Anterior roots.

Chart showing the sensory tracts in the spinal cord with entrance of the sensory nerve-roots.

PLATE IX.

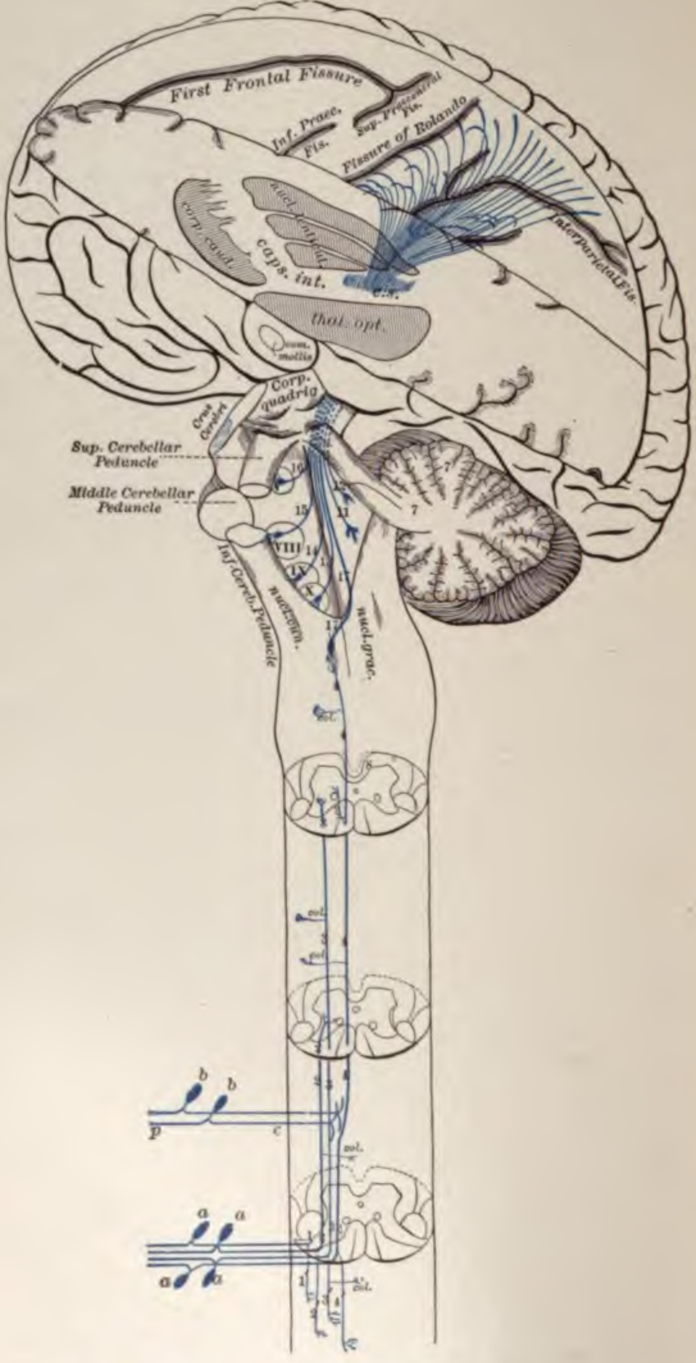


Diagram showing Course of Sensory Fibres from Periphery to Cord, Cerebrum, and Cerebellum. (Flatau.)

hand, a lesion of any size in this region will usually produce signs of motor paralysis, owing to the juxtaposition of the motor and sensory fibres. As yet the full diagnostic value of changes in the stereognostic sense has not been determined.

Disturbances in the sensation of the skin may arise from functional or organic disease involving the peripheral nerves, the sensory tracts in the spinal cord, similar tracts in the lower part of the brain, and, finally, the subcortical or cortical parts of the cerebrum itself.

The sensory pathway or the afferent fibres pass upward, starting with the peripheral sense-organ in the skin, or elsewhere, and after forming part of the nerve trunk and entering the ganglion on the posterior root, enter the spinal cord by what is called the posterior root, which is shown in Fig. 75 near the words "Lissauer's zone." (See Figs. 74 and 75.)

The posterior root enters the cord in three sets of fibres; one of these, the one lying nearest the posterior median fissure, is composed of coarse fibres and is called the median bundle, and passes obliquely into the lateral part of the column of Burdach.

Some of these fibres also enter the column of Goll. As soon as they have entered this column they turn at right angles and run upward for some distance, thereby helping to form the column of Burdach. Some of them also run downward a short distance.

The second set, near the side of the cord, goes directly into the gray matter of the posterior horn through the substance of Rolando, and the third set, nearest the side of the cord, enters the cord very superficially, and, turning at once at a right angle, goes upward to form Lissauer's zone. The longitudinal course of these fibres is shown in Plate IX. Here it is seen that they pass upward chiefly in the column of Goll (posterior median) to the medulla oblongata. Before reaching the medulla, however, the column of Goll ends in the gracile nucleus and the column of Burdach in the cuneate nucleus.

These nuclei which have received the fibres of the two sensory columns give origin to fibres which pass to the brain. They sweep forward to the front of the central canal of the medulla and decussate at a higher level than the motor tracts. A great majority of these fibres pass upward to the brain, but some pass forward, and finally join the restiform body on the posterior aspect of the medulla. Those which pass upward from the so-called fillet pass into the crus cerebri, in that part of it called the tegmentum, and thence into the posterior part of the posterior limb of the internal capsule, whence they spread out in the corona radiata to the occipital lobe and temporo-sphenoidal lobes.

The duty of the physician in all cases is to determine first whether the disorder of sensation is functional or organic, and then where the lesion producing the symptoms is situated.

The two chief manifestations of perverted sensibility in the skin are anæsthesia and hyperæsthesia, and the minor ones are paræsthesia or numbness, tingling and formication, and analgesia, or the failure to feel pain. Whatever the cause of these symptoms may be, the history of the patient and his general symptoms should be carefully studied when examining these signs, as frequently a diagnosis is impossible with them alone as guides.

Anæsthesia. Anæsthesia of the skin is indicative of a very large number of conditions arising anywhere in the sensory apparatus. In other words, anything which interferes with the transmission of an impulse to the perceptive centres in the brain may be its cause. Of the functional causes, the most frequent is hysteria, and the presence of cutaneous anæsthesia in a female should always arouse a suspicion of its being due to this cause. Rarely it is seen in hysterical males. The organic causes of anæsthesia of the skin are cerebral hemorrhage, cerebral tumor, hemorrhage in the pons or tumor of the pons, hemorrhage in the cord, tumor of the cord, myelitis (transverse), locomotor ataxia, cerebro-spinal meningitis, spinal meningitis; compression of the cord by vertebral caries, by fractures, by dislocations; and hemorrhage into its membranes. Additional causes are pressure on the posterior nerve-roots by reason of caries and growths, inflammation of the nerves (neuritis), injuries to the nerves by blows, pressure, or cutting, and, finally, by paralysis of the nerve-endings from cold or the action of drugs.

Anæsthesia, according to its area of distribution, may be divided into hemianæsthesia, crossed anæsthesia, bilateral anæsthesia, irregular but complete anæsthesia, and partial anæsthesia.

HEMIANÆSTHESIA occurs most frequently as a result of hysteria, next commonly from lesion of the posterior part of the internal capsule, and more rarely from spinal injuries or growths in the cord of a unilateral character.

The hemianæsthesia of hysteria involves, as its name implies, one side of the body, and is usually universal on that side, except that here and there may be patches of hyperæsthesia or tenderness, dotted like oases in the midst of the absence of sensation. This anæsthesia is often unaccompanied by motor paralysis, and its area is separated from the opposite side of the body by a sharp line of demarcation, which runs along the middle of the trunk and face. The presence of such a well-defined line of separation in a young woman is of great significance. The anæsthesia is generally absolute, and severe injury may be done to the skin in some cases without the patient feeling it; but, notwithstanding its degree, it is a noteworthy fact that the anæsthesia may transfer itself to the opposite side of the body with great suddenness, and equally suddenly return to its former site. In a great majority of cases, for some unexplained reason, the left side is the one affected by anæsthesia, and hyperæsthesia on the opposite side increases the contrast which exists between it and that in which sensation is lost. (See Hyperæsthesia.) In some cases of hysterical hemianæsthesia the paralysis of sensation involves the nerves of special sense; and loss of smell, taste, and hearing, and impairment of sight may ensue on the same side. The visual changes are so characteristic that they practically decide the character of the case when they are discovered in any instance of doubtful diagnosis; they consist in a loss of the color-vision (first, violet is lost, then blue, and then red), and there is a great limitation of the visual field, whereas in the hemianæsthesia due to an organic lesion in the internal capsule, so situated as to involve the nerve-fibres connected with vision, there is hemiopia. Hemianopsia due to hysteria is so rare as to be denied an existence by most authorities, but Lloyd and de Schweinitz have seen a case. Generally the loss of vision on the anæsthetic side is a total one for both sides of the eye in hysterical blindness. (See chapter on Eye.) Nearly always in hysterical hemianæsthesia a spot can be found over the shoulder which is not anæsthetic. The age of the patient, her sex, the general expression of the face, and the history of her illness, associated, as is frequently the case, with some or all of the hysterical symptoms detailed further on in this chapter, will generally decide the diagnosis in favor of hysteria.

A form of hysterical hemianæsthesia very apt to lead to an error in diagnosis is that seen in persons who have suffered from infantile cerebral paralysis with the resulting deformity (a disease not characterized by sensory disturbances), but who have in later life, superimposed upon the old picture of disease, that of hysteria with this sensory manifestation.

Anæsthesia irregular in its distribution, or absolute hemianæsthesia, may occur in the course of chorea. The presence of chorea in its motor manifestations clears up the diagnosis as to the cause of the loss of sensation.

Hemianæsthesia when not hysterical is nearly always due to an organic lesion in the posterior part of the hinder limb of the internal capsule on the opposite side of the brain from the anæsthesia, and the additional symptoms which sometimes accompany it depend for their existence upon whether the lesion is large enough to involve, not only the fibres from the cutaneous areas, but also those of special sense, such as sight, hearing, or taste. Nearly always the area destroyed is sufficiently large to result not only in hemianæsthesia, but also in loss of motion on the same side. The loss of sensation in such a case is rarely as complete as in hysteria, and the sole of the foot and palm of the hand are often not affected. In rare instances, however, the hemianæsthesia of capsular disease may be absolute and universal, or, more rarely still, occur in patches, thereby closely resembling the anæsthetic areas seen in hysteria.

Hemianæsthesia may also be produced by a large lesion of the cortex in the occipital, temporal, and parietal lobes, in which case it will involve the side of the head as well as the trunk, and will be associated with such definite evidences of apoplexy or injury that the diagnosis will be readily made. If it is widespread, all the special senses will be involved.

Sensory disturbances of the skin are more frequent in softening of the brain than in hemorrhage into the brain, and most commonly are associated with subcortical, rather than cortical lesions.

In this connection it should be remembered that the irregularity of distribution of the lesions in disseminated sclerosis may cause a hemianæsthesia, partial or complete.

Anæsthesia resulting from tumor of the brain occurs in about 20 per cent. of the cases, and may be unilateral and confined to the paralyzed side, or appear as an isolated symptom without motor paralysis. When of the latter form it is often associated with lesions in the neighborhood of the fissure of Rolando, and in tumors involving the posterior parietal region and the posterior part of the internal capsule.

Autopsies and experiments show that hemianæsthesia may arise from a lesion in the optic thalamus, but such an occurrence is very rare.

A very important and essential factor in making the diagnosis that the anæsthesia is cerebral in origin is the history of the beginning of the attack, which has been sudden if due to hemorrhage, embolus, or thrombus (see Hemiplegia), and characteristic of the condition which we call apoplexy.

Unilateral anæsthesia associated with motor paralysis, both being somewhat irregular in their distribution, may be due to a lesion, such as a tumor in the pons or medulla oblongata, but death so commonly ensues soon after the apoplexy that the symptom is often overlooked or cannot be developed. Further, the discovery of such anæsthesia does not positively localize the lesion in the pons, for we do not know much about the course of the sensory fibres in this part. If, however, the area supplied by the trifacial nerve, namely, the face, is anæsthetic, and these symptoms are associated with it, then it is fair to assume that the trouble lies in the pons and has involved the nucleus of the fifth nerve. (See Anæsthesia of Face.)

Anæsthesia of irregular distribution or confined to one limb may result from cerebral or spinal lesions, or be due to a neuritis, of which we shall speak further on. If it is a mono-anæsthesia from cerebral disease, which is very rare, the anæsthesia is most marked at the distal part, and gradually fades off as the trunk is approached. It is evenly distributed, so far as circumference is concerned, and has no sharp line of demarcation.

When such an anæsthesia is due to spinal disease the cause may be tumor of the spinal cord, the symptoms depending in their character on the area involved; but in any event the upper border of the area involved is sharply outlined and a constriction-band sensation is often present.

The irregularly distributed form of anæsthesia due to hysteria has the same general peculiarities of migration as are seen in hemianæsthesia from this cause, and in its symmetrical form it closely resembles the anæsthesia due to multiple neuritis. Thus, in the hand the area of anæsthesia may be that covered by a gauntlet glove, in the foot that covered ordinarily by a sock, the line of normal sensation being present just above the place to which these protections usually extend.

CROSSED ANÆSTHESIA. When sensory paralysis of one side, associated with partial paralysis of motion or paresis on the same side, comes on, and with it there is hyperæmia of the skin on that side from vasomotor paralysis, there is a strong probability that there is a lesion in the cerebral peduncle of the opposite side. If there is at the same time paralysis of the muscles supplied by the oculomotor nerve on the opposite side from the anæsthesia—that is, on the same side as the lesion, this diagnosis is still further confirmed; and if the tongue and half of the face on the anæsthetic side of the body are paralyzed, still further confirmatory evidence of a peduncular lesion is obtained. Thus there might be hemianæsthesia and paralysis of the right side of the body, including the face and right half of the tongue, and ptosis, from oculomotor palsy, on the left side of the face. The paralysis of the body, face, and tongue would be on the side opposite to the lesion, but the oculomotor paralysis would be on the same side as the lesion.

Crossed anæsthesia of the limbs and face—that is, anæsthesia of one side of the body with anæsthesia of the opposite side of the face—can only occur in lesions involving the upper part of the pons in such a way that the fibres of the trifacial are diseased on one side, and the path for sensory impulses of the other side of the body is also destroyed. (See chapters on Face and Head, and on Hemiplegia.)

An important point to be noted in the diagnosis of cerebral anæsthesia is the fact that the reflexes are preserved, though the patient may not feel the touch or painful impression; that is to say, irritation of the skin causes movement in the arm or leg, not by any intention of the patient, but owing to the fact that the sensory centres in the cord receiving an impulse cause the corresponding motor centres to send out impulses which contract the muscles.

Partial hemianæsthesia, with partial hemiplegia on the opposite side in crossed paralysis, may occur from lesions on one side of the spinal cord, and if high up, involve a large part of the trunk and lower limbs. (See chapter on Feet and Legs, part on Myelitis.) These cases have been explained by a theory of Brown-Séquard, which has recently been doubted owing to the studies of Mott and others. Thus, until recently, it was considered as proved that sensory impulses entering the cord crossed to the opposite side almost at once, at least in greater part, passing to the lateral columns in front of the pyramidal tract, and that a very small number entered the posterior columns, while a few ascended in the gray matter. The recent studies of Mott, in confirmation and criticism of still other investigators, seem to prove that the reverse is the case, and that the greater part of the sensory impulses do not cross the cord, only a few fibres passing to the opposite side on entrance. He believes that the main pathway for heat and cold sensations is in the gray matter, while the tactile pathways are in the posterior columns, although it is possible that some few isolated fibres may exist in the lateral columns and that these cross in the cord about the level of entrance.

BILATERAL ANÆSTHESIA. Anæsthesia of hysterical origin involving both legs, and sometimes the lower part of the trunk on both sides, may occur, and, aside from the typical signs of hysteria in general which distinguish it, may be discovered by the fact that in hysteria the failure of sensation does not involve the skin of the genitals, as it does in organic lesions producing somewhat similar symptoms. In addition it will be found that in hysteria a V-shaped piece of skin over the sacrum is not anæsthetic. Anæsthesia of this variety, corresponding in the sensory organs to what we call paraplegia in the motor apparatus, is practically never produced by a cerebral lesion, and, if not hysterical in cause, must be spinal; but it is much more rare than is motor paralysis in these parts from lesions in the spine. When it does ensue from spinal causes motor paralysis will in the great majority of cases be found associated with it at least to some extent. To express it concisely, the characteristic of a typical spinal anæsthesia is that it is bilateral and usually involves both sides quite symmetrically; that motor paralysis is generally associated with it; that the reflexes are greatly perverted; and that trophic changes may be present as a result of an involvement of the trophic cells in the anterior cornua coincidently with the disease of the sensory parts of the cord.

The diseased conditions of the cord which result in symmetrical anæsthesia of the skin of the legs and trunk are, first and most prominent, locomotor ataxia; second, myelitis; and, finally, hemorrhages, tumor of the cord or its membranes, meningitis, or injuries which cause pressure on the sensory tracts by producing fracture of the vertebræ or dislocation. Very rarely, however, a lesion of the pons may so result.

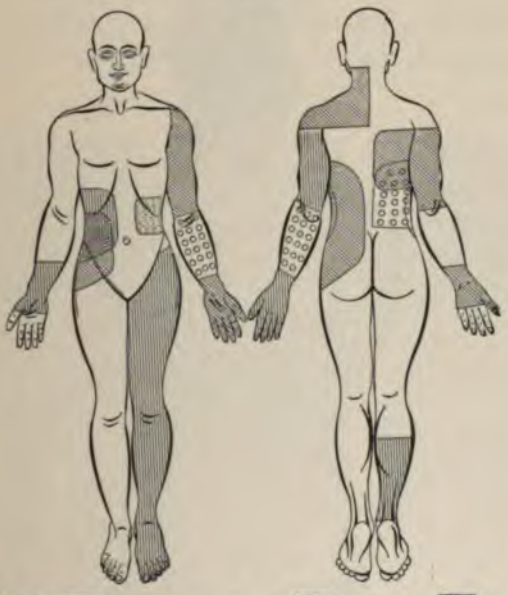
Anæsthesia of the lower portions of the body and legs occurs in the later stages of locomotor ataxia, and is usually preceded by forms of paræsthesia. (See Paræsthesia.) The anæsthetic areas are most marked in the soles of the feet and about the malleoli, according to Belmont. In other words, blunting of sensibility is seen in nearly all cases of tabes dorsalis late in the disease. In some cases the sense of touch is preserved and the sense of pain lost (analgesia), while in others the opposite condition is present. Again, we find loss of tactile sense and of pain-sense without loss of heat and cold sense, and *vice versa*. A very characteristic sensory symptom of tabes is the delay in the recognition of an irritation of the sensory nerves, so that if the patient be blindfolded and then pricked with a pin he will not make an exclamation or draw his foot away for several seconds. In other instances the patient complains of repeated pricks when only one has been given, or, when asked the number of points pricking him, states that there are four or five instead of the one really present. If, in addition to these sensory disturbances, we find Romberg's symptom (see Legs), Argyll-Robertson pupils (see Eye), and loss of patellar reflex (see Reflexes), and a number of other diagnostic peculiarities of tabes, the decision as to the cause of the anæsthesia is easily made.

Slight anæsthesia, retardation of the transmission of sensory impulses from the skin, and perversion of temperature-sense may be rarely developed late in the course of Friedreich's ataxia.

Bilateral anæsthesia of the character just discussed, as caused by locomotor ataxia, may also occur as a result of acute or chronic myelitis. The first change under these circumstances is a mere obtunding of sensitiveness, which gradually deepens till loss of pain-sense, pressure-sense, and, lastly, complete anæsthesia is developed. The development of these symptoms indicates involvement of the posterior columns. Loss of reflex activity in the legs is developed in direct proportion to the destruction of the motor and sensory nerve-tracts in the cord. The predominance of motor paralysis, the fact that the lower limbs are both involved, and the absence of the characteristic symptoms of locomotor ataxia all tend to make the diagnosis certain, while the absence of the pains of tabes and of the other signs of that disease still further excludes its presence from the case. Further than this, the myelitis creeps up the cord, involving new areas, and new parts of the skin become anæsthetic. An important point, too, in regard to the anæsthesia of acute myelitis is this, namely, that, while in the upper extremities the loss of sensation and motion is associated, so that both functions are lost in the same area, in the lower extremities these two functions are not lost in the same areas. Thus myelitis of the lumbar enlargement in its lower part is accompanied by anæsthesia of the gluteal area and motor paralysis of the anal muscles; and, again, anæsthesia of the gluteal region, the back of the thigh, and the back of the calf is associated with loss of power in the muscles that move the foot, while in lesions of the upper part of the lumbar segment the anæsthesia involves the thigh, the inner side of the leg, and the foot, in association with paralysis of the quadriceps extensor and deeper muscles of the thigh. (See chapter on Feet and Legs, part on Myelitis.)

The development of sudden bilateral anæsthesia, which is accompanied by severe pains of a tearing or burning character, creeping rapidly up the body, is indicative of acute hemorrhage into the spinal membranes, or it may be due to that very rare lesion, hemorrhage in the cord. In either case motor paralysis is present. Anæsthesia, or the milder perversions of normal sensibility of the skin, may be present in cases of compression of the cord by caries, and by spinal curvature, tumors, or aneurisms producing erosion. Sometimes, while tactile anæsthesia is complete in these cases, severe pain is constantly suffered (anæsthesia dolorosa), and this is often the case, according to Wood, in cancer of the spine.

FIG. 76.



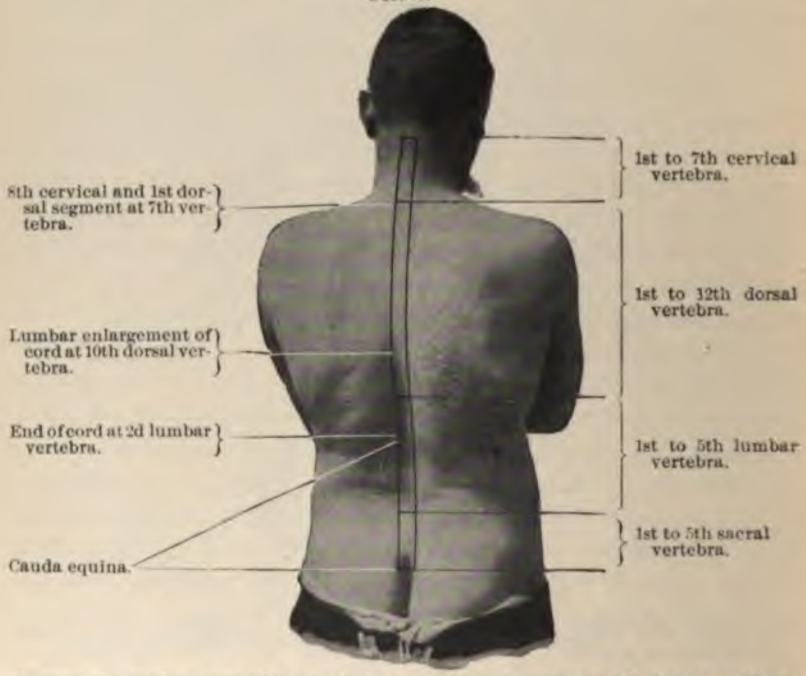
Sensory chart, showing areas of . . . . . ▨ *Thermo-Anaesthesia,* ▥ *Analgesia,* ▩ *Thermo-Anaesthesia and Analgesia* ▒ ***Tactile Anaesthesia***, and areas in which the patient's answer to tests of temperature showed reversal ⁝ *Cold-Hot; Hot-Cold,* in a case of syringomyelia. (DERCUM.)

Partial anæsthesia of the skin of the trunk and arms of a bilateral character, associated with progressive muscular atrophy, scoliosis, and trophic lesions in the skin, points strongly to syringomyelia.

The loss of pain- and temperature-sense is usually the first symptom. The areas of anæsthesia are best shown in Fig. 76.

Having considered the general spinal causes of anæsthesia of the skin, it yet remains to determine what part of the cord is involved by the pathological process; and this is, fortunately, possible, chiefly through the very accurate and noteworthy studies of M. Allen Starr, Thorburn, and Head, not to mention collateral ones of great value by Horsley and many others; but the field is only partly covered, and some of our uncertainties depend upon lack of knowledge as to the course of the sensory fibres in the cord.

FIG. 77.


8th cervical and 1st dorsal segment at 7th vertebra.
Lumbar enlargement of cord at 10th dorsal vertebra.
End of cord at 2d lumbar vertebra.
Cauda equina.
1st to 7th cervical vertebra.
1st to 12th dorsal vertebra.
1st to 5th lumbar vertebra.
1st to 5th sacral vertebra.

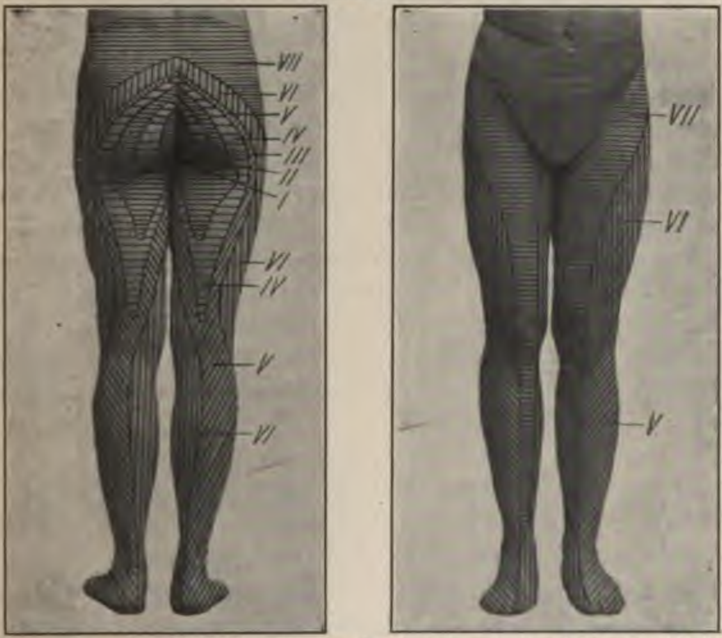
Showing the surface-areas of the back corresponding approximately to the areas of the spinal cord supplying the trunk and limbs.

Roughly, we may state that disease of the cervical cord generally produces disturbances of sensation in the arms, hand, and fingers; disease of the dorsal cord, disturbances in the sensation of the back and trunk, which may radiate into the thighs; and disease of the lumbar cord gives rise to these symptoms in the legs and feet.

Again, it is to be remembered that, as a rule, in a transverse lesion of the spinal cord the anæsthesia begins at a level which is three or four inches below the lesion in the cord (Horsley and Gowers); this being due, as proved by Sherrington, to the fact that each area of skin is supplied by three nerve-roots whose peripheral filaments overlap one another.

For the ready study of the subject the cord has been separated into segments corresponding with the vertebræ covering it. The areas of anæsthesia produced by spinal injury or disease are best described by Starr's well-known article and diagrams, from which we quote. In this connection the reader should refer to the tables on pages 121 and 122, showing the localization of the functions of the segments of the spinal cord. (See chapter on Legs and Feet.)

FIG. 78.



Areas of anæsthesia in lesions at various levels of the spinal cord from sacral V. to lumbar II. (After STARR.)

I. Sacral V.
II. Sacral IV.
III. Sacral III.
IV. Sacral I.
V. Lumbar V.
VI. Lumbar III.
VII. Lumbar II.

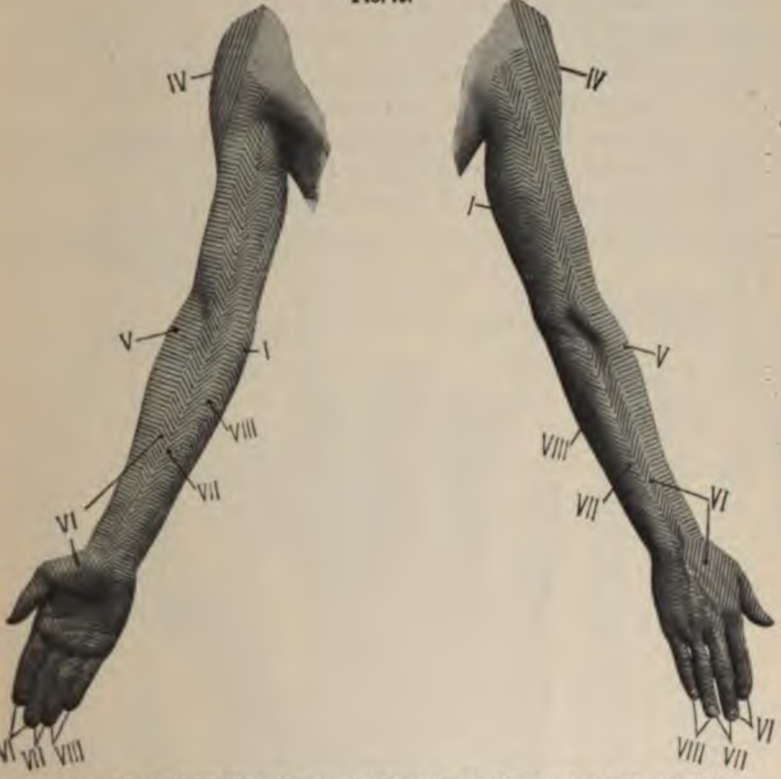
The anæsthetic areas included in zones I. and II. in Fig. 78 are due to a lesion involving the conus medullaris and the fourth and fifth sacral segments of the cord. These zones include the peritoneum, the posterior part of the scrotum in males, the vagina in females, and the mucous membrane of the rectum. Anæsthesia in zone III. is due to lesion of the third, fourth, and fifth sacral segments, and includes a large part of the buttock and the upper part of the thigh, posteriorly, in a triangular space. Zone IV. is practically an enlargement of zone III. in every direction, particularly toward the popliteal spaces, and is probably due to a lesion in the first and second sacral segments; but this needs confirmation by autopsy, as Starr points out. Zone V. includes all the first four zones just named, and extends down through the popliteal space in a band-like shape; after it passes this space it descends the outer side of the leg and foot, sometimes ending at the ankle, sometimes at the sole or the three outer toes and half the next toe. Such an area indicates a lesion involving all the segments of the sacral cord, and extending into the lumbar cord to the fifth lumbar segment. Zone VI. is caused by a lesion extending to the third lumbar segment, and when it is present the anæsthesia covers the back of the thighs and legs and also the front of the thighs, except in an area which extends from above downward along the shin, sometimes to the foot, as in Fig. 78. If the foot is involved, the lesion in the lumbar cord is probably above the third lumbar segment. Zone VII., which is larger than all, follows a lesion in one of the four lumbar segments—that is, all but the first. The line of anæsthesia, Starr tells us, is lower in front than behind. When the abdominal wall is involved in the anæsthesia the first lumbar segment is probably diseased.

The area of the anæsthesia from the level of the abdomen corresponds very closely to the levels in the cord if we allow for the space already mentioned, of two to three inches for the interlacing anastomosis of the nerve-fibres of the posterior roots.

They are about as follows, according to Thorburn: when the anæsthesia is as high as the anterior inferior spine of the ilium, the lesion is at the twelfth dorsal vertebra; if at the umbilicus, at the eleventh and twelfth dorsal vertebræ; if up to the lowest floating rib, the whole eleventh dorsal vertebra; if from one to four inches above the umbilicus, the ninth and tenth dorsal, and perhaps part of the eighth dorsal vertebra; if as high as the nipples, the fourth dorsal vertebra, and if to the third rib, the lesion is as high as the second dorsal vertebra.

Starr has also given us, in another paper than that already quoted, equally good ideas of the areas of anæsthesia occurring above those just described. (Fig. 79.) When the anæsthesia extends to the arms and is found upon the inner side of the arm and forearm, reaching to the wrist, but not to the hand, and also involves a small zone on the extensor and flexor surfaces of the arm and forearm, the second dorsal region is the site of the lesion. If the anæsthetic area includes the ulnar side of the hand, the palmar and dorsal surfaces of the same, and the little finger, and extends in a narrow strip up to the axilla on both the anterior and posterior surfaces of the arm and forearm, the lesion is probably at the level of the eighth cervical vertebra. When the zone involved extends to the middle of the central figure on the palmar and dorsal aspects, and runs up

FIG. 79.

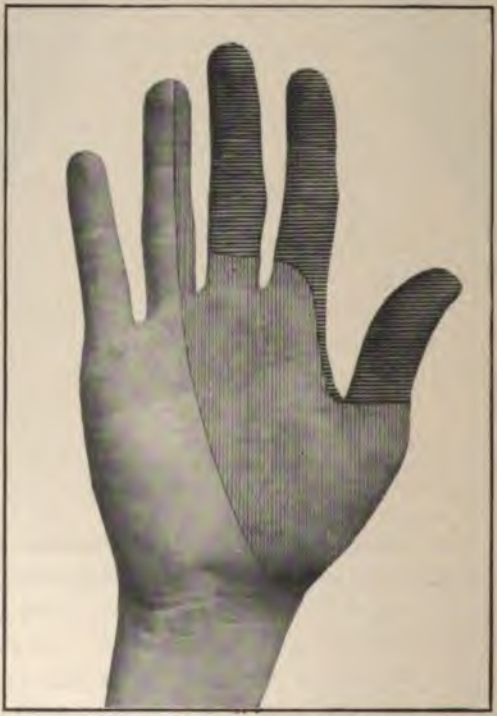


Areas of anæsthesia from lesions at various levels of the spinal cord from the second dorsal to the fifth cervical. (After STARR.)

the centre of the forearm and arm, the seventh cervical area is diseased. Again, when the remaining skin of the hand up to the wrist and a narrow strip of skin up the forearm and arm on both surfaces to the axilla is affected, the lesion is at the sixth cervical vertebra, while anæsthesia of the forearm and arm on the outer surface as high as the deltoid insertion indicates that the fifth cervical vertebral area is in trouble. Lesions higher than this usually produce death before it is possible to test sensibility.

In order that the reader may gain a still better idea of the probable, or rather approximate, area of distribution of the spinal nerves, the three charts of Thorburn, Starr, and Head are here reproduced, as prepared in colors, with significant lettering by Thorburn, for the *International Medical Annual* for 1896. (Plates X., XI., and XII.)

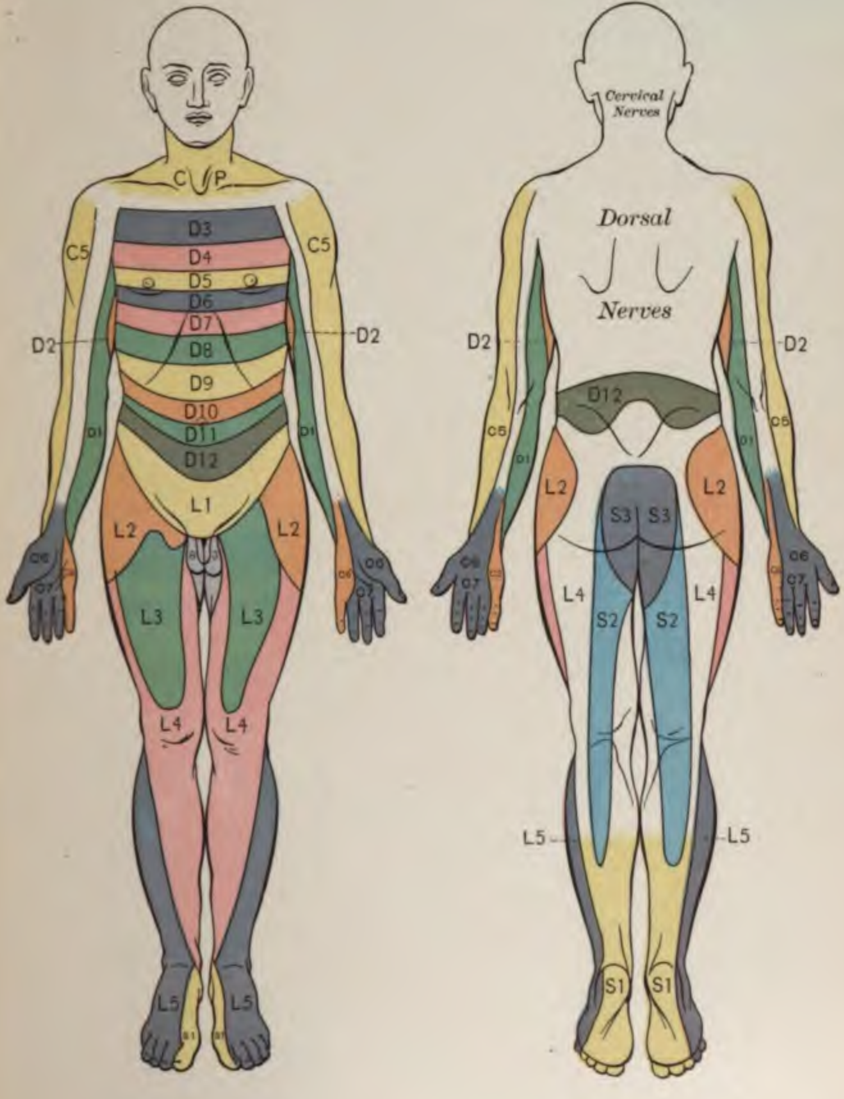
FIG. 80.



Area of anæsthesia from injury of the median nerve. Palmar surface.

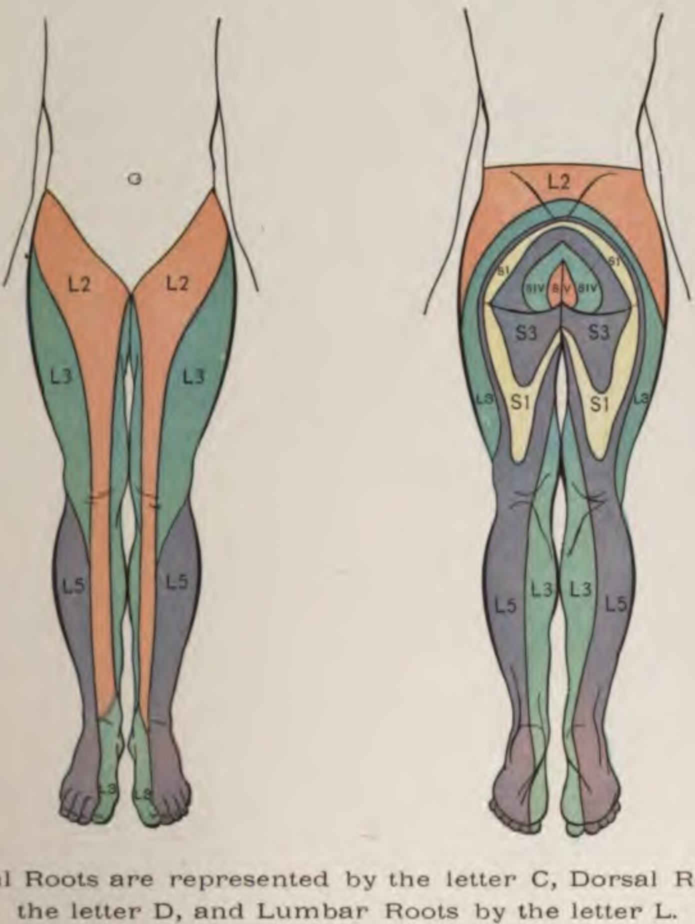
Neuritis as a Cause of Anæsthesia. Anæsthesia of the skin

PLATE X.



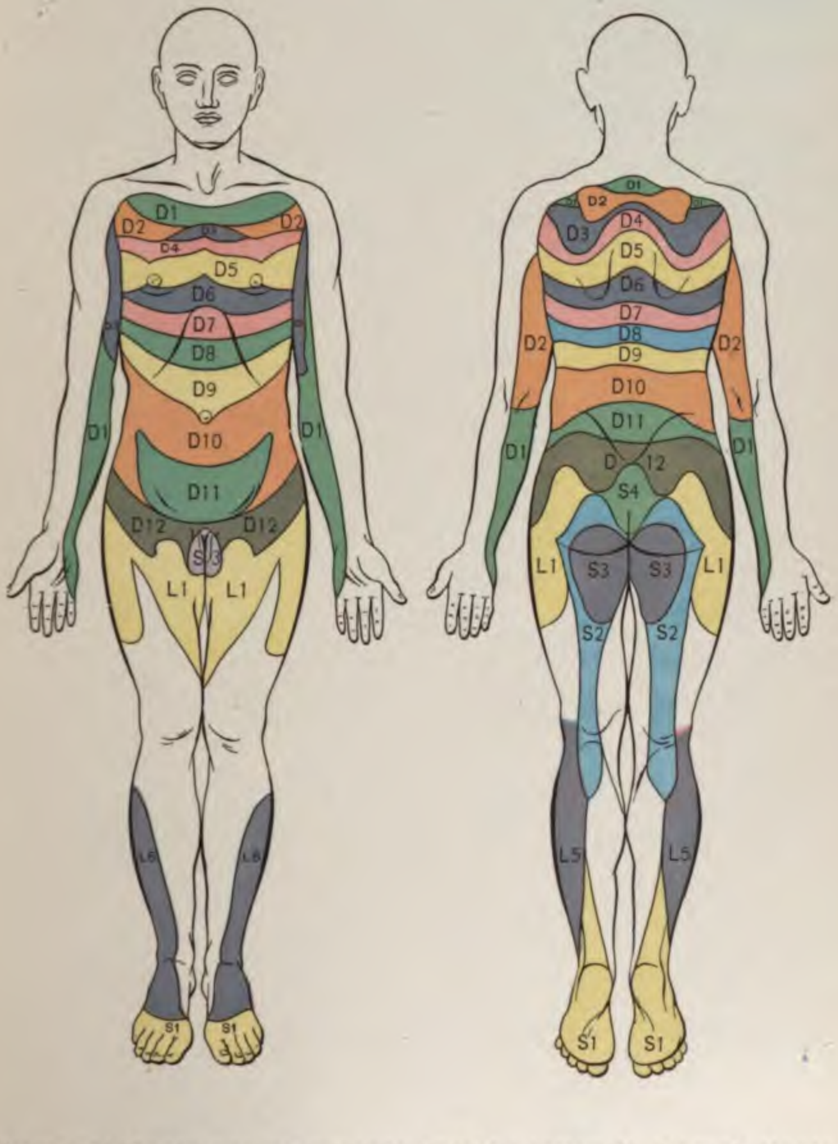
Cervical Roots are represented by the letter C, Dorsal Roots by the letter D, and Lumbar Roots by the letter L. (Chart after Thorburn.)

PLATE XI.



Cervical Roots are represented by the letter C, Dorsal Roots by the letter D, and Lumbar Roots by the letter L.
(Chart after Starr.)

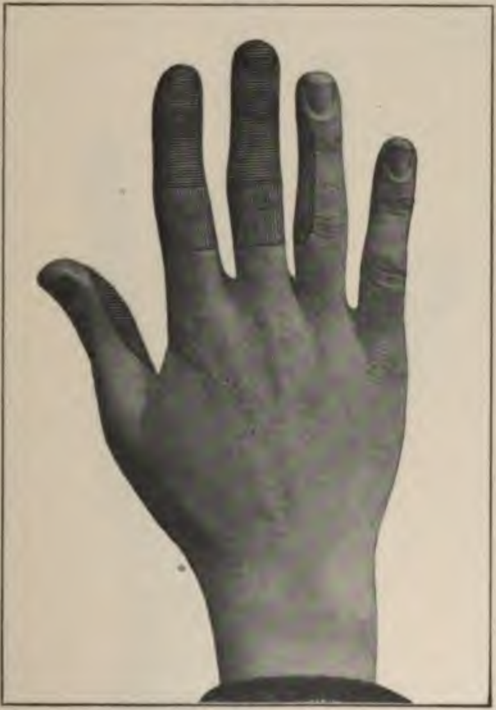
PLATE XII.



Cervical Roots are represented by the letter C, Dorsal Roots by the letter D, and Lumbar Roots by the letter L.
(Chart after Head.)

in any part of the body may be due not only to cerebral or spinal lesions, but also to neuritis or inflammation of the nerve-trunk, or to some injury which impairs its functional activity by pressure, bruising, or cutting. As a rule, loss of sensation from neuritis occurs late in the disease, hyperæsthesia or paræsthesia being the earlier manifestations; but in some cases these are absent, and anæsthesia begins at once. The characteristic of such an anæsthesia is that it is confined to the area supplied by the affected nerve, although the presence of a multiple neuritis may produce such a universal anæsthesia by involving all the nerves that this sign is masked. While a mono-anæsthesia may be due to other causes, it is in the great majority of cases due to neuritis. The signs of an anæsthesia due to neuritis are loss of motion and sensation, tenderness on pressure over the nerve-trunks supplying the affected area, trophic changes in the tissues of the part, with the development of reac-

FIG. 81.

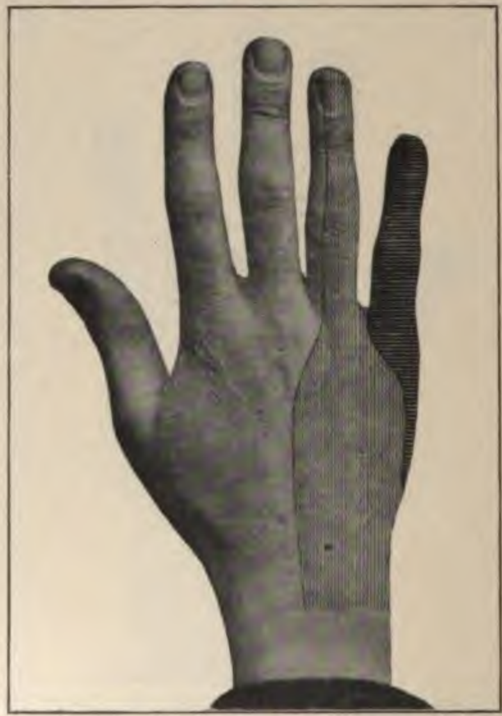


Area of anæsthesia from injury of the median nerve. Dorsal surface.

tions of degeneration and pain in the involved nerves or parts supplied by them. Somewhat similar symptoms occur in anterior poliomyelitis, but pain is not commonly present in this disease, and there is no anæsthesia, either in children or adults. (See chapters on Hands and Arms, and on Feet and Legs.)

Toxic peripheral neuritis producing anæsthesia may arise from poisoning by arsenic, lead, alcohol, or mercury, from septic states of the body, and from the infectious diseases, particularly diphtheria, influenza, and typhoid fever.

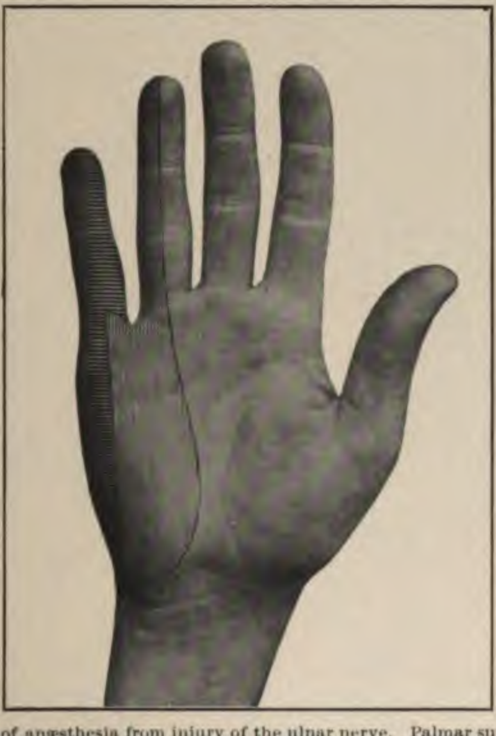
FIG. 82.



Area of anæsthesia in injury of the ulnar nerve. Dorsal surface.

That due to the mineral poisons has in each case certain differential points of importance. The anæsthesia of arsenical poisoning is more marked than in lead-poisoning, in which condition it is often almost absent, and the lower extremities are very apt to be involved, whereas in lead-poisoning, as is well known, the nerves of the arm are particularly susceptible. (See Arms and Hands.) Arsenical neuritis may also produce pigmentation of the skin. In alcoholic neuritis the temperature of the anæsthetic areas is often subnormal and there are nearly always mental disturbances represented by delusions. In mercurial poisoning, shaking like paralysis agitans may be present. An analysis of the motor symptoms in all these cases is important, and the discovery of any one of these poisons in the urine, with the history of the patient, generally makes the diagnosis possible.

FIG. 83



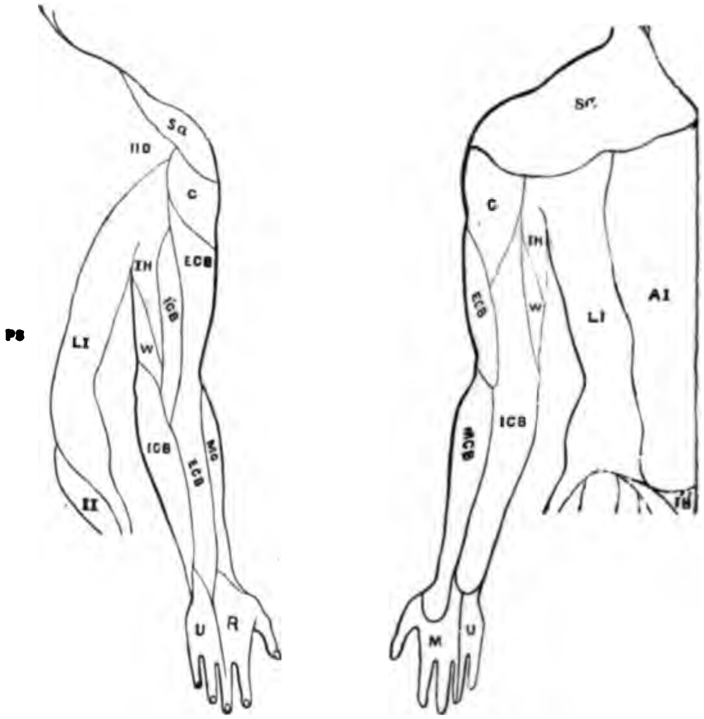
Area of anæsthesia from injury of the ulnar nerve. Palmar surface.

Diphtheritic neuritis is quite common, and in 50 per cent. of the cases in which it occurs sensibility is lost or disturbed in the areas supplied by the involved nerves.

Great care is needed in all cases of neuritis lest the mistake be made of diagnosing the condition as one of locomotor ataxia, when in reality it is pseudo-tabes.

It has already been stated that in neuritis the area of anæsthesia is that supplied by the affected nerve. For this reason we can determine what nerve-trunk is affected by studying the area of anæsthesia, always remembering, however, that the sensory fibres of the nerves, particularly in the hands and feet, anastomose so freely with those of adjacent nerves that the area of the anæsthesia may not be exactly that supplied by the nerve involved; or, in other words, the presence of loss of power in a region supplied in health by a nerve which has been divided is constant, but very often sensation is not disturbed, even though the divided nerve be the sensory as well as the motor supply to the part.

FIG. 84.

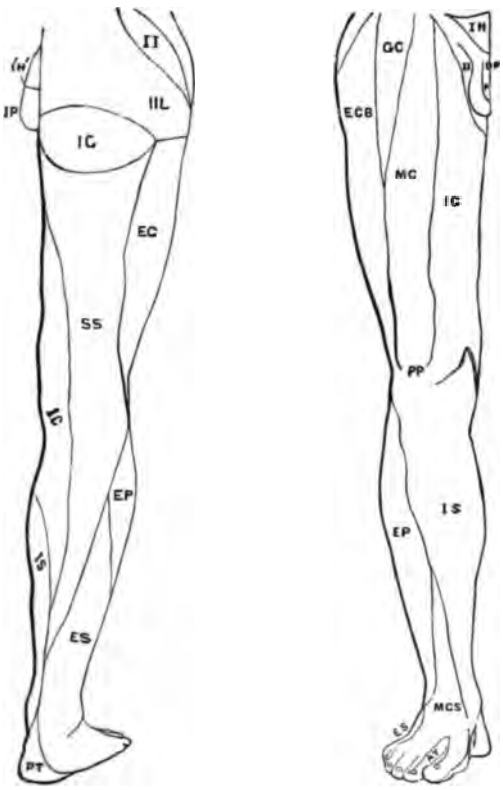


Cutaneous nerve-supply of the trunk and upper extremity. (FOWLER.) SC. Supraclavicular nerve. IID. Second dorsal. PS. Posterior branches of the spinal nerves. LI. Lateral branches of the intercostal nerves. AI. Anterior branches of the intercostal nerves. C. Circumflex nerve. IH. Intercostal humeral. W. Nerve of Wrisberg. I'CB. Internal cutaneous branch of musculo-spiral nerve. ECB. External cutaneous branch of musculo-spiral nerve. ICB. Internal cutaneous nerve. MC. Musculo-cutaneous nerve. R. Radial nerve. U. Ulnar nerve. M. Median nerve.

It is well to remember also that sensory disturbances of the skin following injuries of nerves are often not nearly so great as the motor disturbance, even where there is no sensory transmission by anastomosis, and where they are present they usually disappear, more rapidly than the motor loss, as recovery takes place.

The following facts are, therefore, of diagnostic interest. If the anæsthesia is found to be due to a neuritis and to involve the palmar surface of the thumb, fore and middle fingers, the median nerve is probably the one at fault (Figs. 80 and 81), and the area may even include in rare instances the backs of these fingers at their

FIG. 85.



Cutaneous nerve-supply of the lower extremity. (FOWLER.) Lumbar plexus. IH. Ilio-hypogastric nerve. II. Ilio-inguinal. IIL. Second lumbar nerve. GC. Genito-crural. EC. External cutaneous. MC. Middle cutaneous. IC. Internal cutaneous. IS. Internal saphenous. PP. Plexus patellæ. Sacral plexus. DP. Dorsalis penis of pudic. IP. Inferior hemorrhoidal of pudic. P. Superficial perineal of pudic and inferior pudendal of small sciatic. IG. Inferior gluteal of small sciatic. SS. Small sciatic. EP. Branches from external popliteal. ES. External saphenous. MCS. Musculo-cutaneous. AT. Branches of anterior tibial. PT. Branches of posterior tibial.

bases and the half of the third finger nearest the thumb. When there is disturbance of sensation in the ulnar side of the ring-finger and in the skin of the little finger, there may be ulnar neuritis (Figs. 82 and 83.) (See also chapter on Hands.) The nerve-supply of the skin of the entire upper extremity is well seen in Fig. 84.

The development of sensory disturbances in the feet, resulting from neuritis, is as follows: When there is perverted sensation of the inner side of the foot from the tip of the big toe to the heel, and thence up the inside of the calf to the knee, the nerve involved is the long or internal saphenous. When the dorsal surface of the foot has its cutaneous sense disturbed the nerve involved is the musculo-cutaneous, a branch of the external popliteal. Disturbance of sensation on the outer side of the foot and calf indicates failure of function in the external saphenous, which is composed of the cutaneous branches of the external and internal popliteal nerves. Disturbed sensation on the posterior surface of the calf also indicates trouble in the external saphenous nerve and communicans peronei, while when the sensation of the skin of the heel is disturbed the plantar cutaneous nerve, a branch of the posterior tibial, is involved. (Fig. 85.)

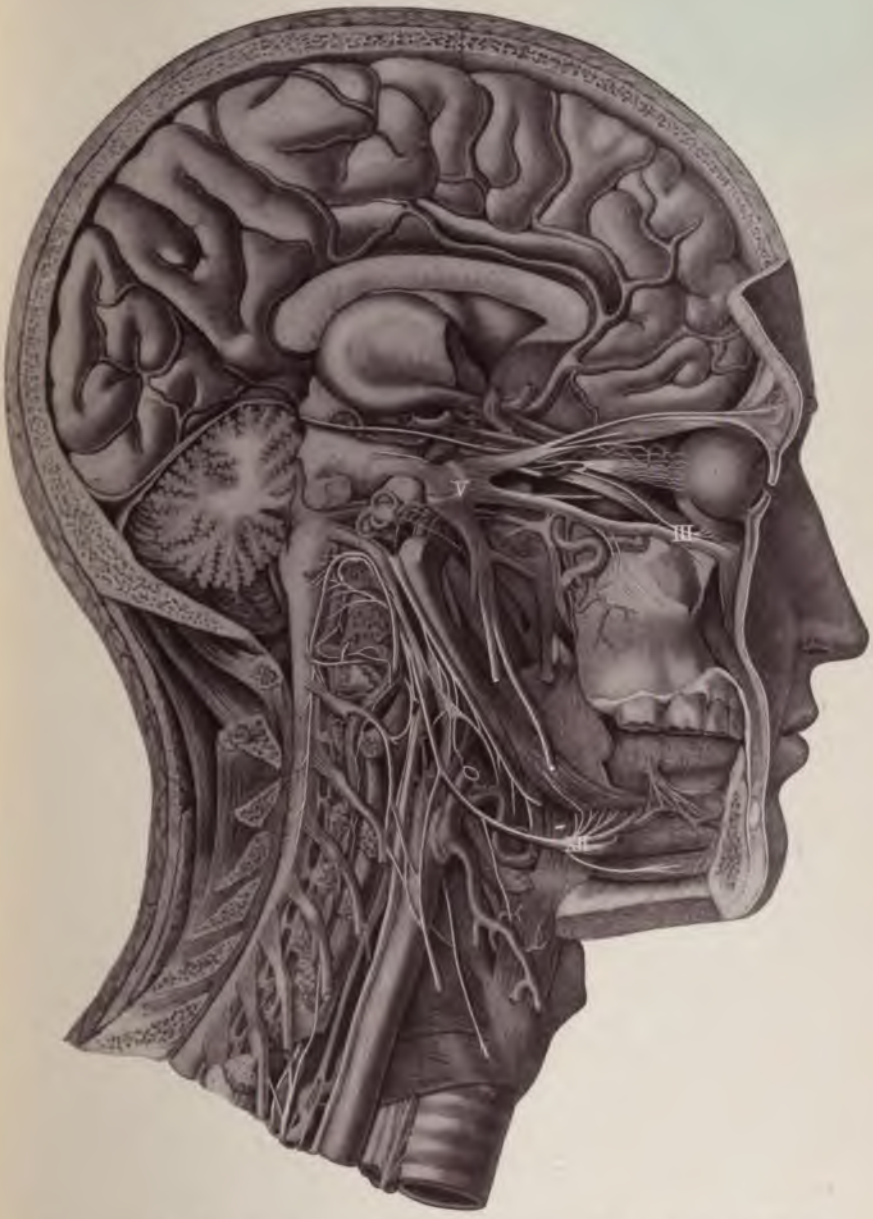
In the skin of the thigh the anterior surface is supplied by the middle cutaneous nerve, which is a branch of the anterior crural; on the inner side by the internal cutaneous, also a branch of the anterior crural; and on the outer side by the external cutaneous, which arises from the second and third lumbar nerves. Laterally the external cutaneous gives the supply. Posteriorly the small sciatic gives the nerve-supply to the skin.

Anæsthesia of the greater portion of the skin of the thigh, except in a narrow strip on the back part and in the area supplied by the internal saphenous nerve, often occurs as the result of paralysis of the anterior crural nerve, arising from pelvic tumors, psoas abscess, and vertebral disease.

Facial anæsthesia and its diagnostic meaning are still to be considered. When it occurs it indicates that the fifth nerve, or its nucleus, is involved.

If the area be that of the forehead, the upper eyelid, the conjunctiva, and the nostril, the ophthalmic branch of the fifth nerve is at fault, and the lesion is probably at the sphenoidal fissure or within the orbit, and reflex winking of the eye no longer takes place because the conjunctiva is anæsthetic.

PLATE XIII.



Showing the Distribution of the Cranial Nerves, Particularly the Fifth.
(Modified from Arnold.)

III. Branch of oculo-motor to inferior oblique.

V. The Gasserian ganglion composed of the fibres of the sensory root of the nerve. The plate would seem to indicate that the three branches of the nerve arise from this ganglion, but they do not, for the motor fibres do not enter the ganglion but join the sensory fibres in the third branch, after they leave the ganglion.

XII. The hypoglossal nerve.

If the skin of the upper part of the face is anæsthetic, the superior maxillary branch is involved; and if the skin of the temporal region and that of the jaw and the under lip are anæsthetic, the inferior maxillary branch is diseased. When both of these branches are paralyzed there is probably a tumor of the superior maxillary bone; and if the entire area of the three branches is anæsthetic, the Gasserian ganglion may be the part affected, and this will be accompanied by trophic changes in the anæsthetic parts. The most common cause of anæsthesia of the trifacial is, however, neuritis.

Romberg makes the following differential statement:

a. The more the anæsthesia is confined to single filaments of the trigeminus, the more peripheral the seat of the cause will be found to be.

b. If the loss of sensation affects a portion of the facial surface, together with the corresponding faucial membrane, the disease may be assumed to involve the sensory fibres of the fifth pair before they separate to be distributed to their respective destinations; in other words, a main division must be affected before or after its passage through the cranium.

c. When the entire sensory tract of the fifth nerve has lost its power, and there are at the same time derangements of the nutritive functions in the affected parts, the Gasserian ganglion, or the nerve in its immediate vicinity, is the seat of the disease.

d. If the anæsthesia of the fifth nerve is complicated with disturbed functions of adjacent cerebral nerves, it may be assumed that the cause is seated at the base of the brain.

Other Disturbances of Sensation than Anæsthesia. The other disturbances of sensation of the skin than anæsthesia, which are usually subjective rather than objective, are paræsthesia, hyperæsthesia, and analgesia.

Paræsthesia—numbness, tingling, or burning—is seen in nearly all cases in which anæsthesia ultimately develops as a result of organic lesions. When a patient complains that he cannot feel the contact of clothing about his feet and legs, or that the feet when he walks feel as if wrapped in some thick material, or as if he were walking on moss, or that the soles of his feet feel as if they were numb and at the same time tickled by ants walking over them, the characteristic sensory disturbance of the skin seen in locomotor ataxia is present.

Often there is tingling or numbness of the fingers, particularly of the ring and little fingers, and a sensation as if a girdle were about the patient is common. These are the subjective disturbances of sensation in tabes dorsalis, and, as they are often the earliest manifestations of the disease, possess great diagnostic importance. The objective sensory perversions consist in the discovery by the physician, when studying the sensibility of the skin, of areas of anæsthesia, analgesia, and hyperæsthesia which are usually bilateral. Belmont has stated that we also find these areas in spinal syphilis, either on one or both sides. Numbness, tingling, and formications affecting the skin are also often early symptoms of brain-tumor in the area supplying the affected part, and this possibility is increased if there is associated spasm. The actual objective sensibility of the skin may be preserved for some time after these symptoms appear, or it may be impaired almost at the outset, owing to the involvement of all or part of the sensory tracts in the cord. Similar symptoms are often seen in the early stages of myelitis. They are very frequently seen after injuries to nerves, and severe tingling in its acute variety occurs when the "funny bone" of the elbow is knocked against an object, owing to bruising the nerve. It is also seen in classes of aconite-poisoning, and when the hands have been exposed to carbolic acid. Paræsthesias are also frequently seen in cases of neurasthenia.

Perversions of sensation in the skin sometimes take a curious form, as, for example, that known as allochiria, in which a sensory impulse in one hand is referred by the patient to the opposite hand. This is seen in tabes dorsalis, myelitis, multiple sclerosis, and hysteria. In other cases, as in paralysis agitans, this perversion takes place in the form of failure to distinguish heat and cold, and subjective sensations of extreme heat are felt. The part affected may actually have its temperature raised several degrees.

Magnan asserts that a sensation as if a worm or bug were crawling under the skin is indicative of cocaine-intoxication.

Very closely associated with the numbness of hysteria or neurasthenia, and lying between functional and organic disease of the nerves, is that condition called acroparæsthesia or waking numbness. This state is usually seen in women past middle life, but may occur in men. On waking in the morning marked formication and numbness of the fingers are present, which usually pass off as the day progresses, but as the condition becomes more marked they may last all day. While there is no anæsthesia, strictly speaking, the disturbed sense of touch renders sewing or performing any small act with the fingers almost impossible. These sensations may be confined to the area of one nerve, as the ulnar, or involve all the skin of the hands, or more rarely of the feet. General nervous excitability is usually associated with the local manifestations. Sometimes the scalp may be the area involved.

Acroparæsthesia is to be separated from the sensory disturbances of hysteria by its irregular outline, for generally in the latter disease the areas are distinctly outlined, by the fact that the hysterical condition is usually unilateral, and by the absence of the characteristic general hysterical symptoms. From organic disease it is separated by the absence of the signs of neuritis about to be described, and by the absence of tenderness, pain, and loss of power. From cerebral or spinal disease it is separated by the absence of symptoms produced by lesions in these parts, and by the facts that in both these lesions there is paralysis of motion in association with the sensory disturbance, and in the case of spinal lesions the symptoms are usually in the legs, while acroparæsthesia generally manifests itself in the hands.

Closely associated with paræsthesia, if not an actual form of it, is the "girdle sensation;" that is, the patient feels as if a tight belt was strapped around a limb or the trunk. This is seen as a prominent symptom in locomotor ataxia, myelitis, and tumors of the cord or its envelopes. When the lesion is in the lower cervical or dorsal region the sensation is in the chest or abdomen; but this relationship between the growth and the sensation of constriction is not always constant. (See chapter on the Feet and Legs.)

Hyperæsthesia of the skin is an important symptom of both hysteria and neurasthenia, and its discovery in association with the peculiar symptoms which occur in the former morbid state confirm a diagnosis most positively. The most important and curious of these hyperæsthesias are the so-called hysterogenous zones, or, in other words, areas involving the skin and subcutaneous parts, which possess great sensitiveness and which, when pressed upon, cause in many cases convulsive seizures of the hysterical type. Not only is this true, but in addition it is a noteworthy fact that after the nervous disturbance produced by this means is set in motion, a second pressure on the hysterogenous zone may arrest the seizure. These zones commonly exist over the ovaries, in the groin, about the periphery of the mammary glands, or upon the spine in the lumbar or dorsal region. (See chapter on Pain.)

The hyperæsthesia due to neurasthenia is to a great extent spinal in character, but the skin of the rest of the back, particularly over the great muscles on each side of the spine, may also be involved. Often the neurasthenic patient or one who has phosphaturia will complain that in brushing or combing the hair pain or extreme sensitiveness is developed upon the scalp, and there may be tender areas on the chest. These areas in neurasthenics can hardly be confused, even by the careless, with the hyperæsthetic zones of hysteria, and the personal history and characteristics of the individual aid still further in separating the two conditions.

Hyperæsthesia of the skin, aside from that seen in hysteria and neurasthenia, occurs in peripheral neuritis and locomotor ataxia, the skin of the back being particularly tender in the latter disease, and the excessive sensitiveness is frequently seen in a zone extending a little above the anæsthetic areas of transverse myelitis, this hyperæsthetic area being soon rendered anæsthetic by the progress of the disease. Hyperæsthesia in the skin of the limbs is also rarely seen in myelitis, and when there is motor paralysis of one side and sensory paralysis of the other it is commonly found on the side on which motion is lost. A condition of excessive dermal hyperæsthesia is also present in cerebro-spinal meningitis, in which disease it is often a very early symptom. It usually appears first in the legs, then in the hands and arms, and, finally, the skin of the face and head become involved.

Hyperæsthesia of the skin occurs, often associated with skin eruptions, in that very rare condition called chronic leptomeningitis. Motor symptoms are nearly always present if the cord becomes involved.

Hyperæsthesia of the skin is considered by some authors to be, when found in association with other characteristic symptoms, almost pathognomonic of brain-tumor. It may be found on the scalp, over a large part of the body, or in the part which is paralyzed. It is also found during the convalescence of typhoid fever and in relapsing fever. It also appears in the paralyzed side of persons suffering from hemiplegia, in the area supplied by a nerve suffering from neuralgia, particularly that of a migraine type, in the scalp of persons suffering from gout, and in the same area in women about the time of the menopause.

General tenderness of the skin or deeper tissues is quite frequently seen in cases of rickets and scurvy, the child crying whenever it is moved, as if sore and tender, and tender spots often appear over the ribs in cases of pleurisy.

Sometimes in a neurotic girl about the time of puberty, or in a woman, one breast becomes exceedingly painful and tender, and the skin of the breast becomes so hyperæsthetic that the slightest touch causes pain. The whole breast is, moreover, tender, and movement of the arm may be impossible, owing to pain thereby caused in the gland. This hysterical breast can be separated from the painful breast due to a tumor by the general diffuse character of the swelling, the failure to outline any distinct mass, the neurotic character of the patient and her age.

The hyperæsthesia of chronic alcoholism may be both dermal and deep, and is well marked along the course of the peripheral nerves, particularly where they emerge from deeper structures. It is also seen in the neuritis of lead- and arsenical poisoning.

Increased sensibility of the skin may follow the use of opium or ergot, and is met with in the course of, or as a sequel of, influenza, and in some cases of profound anæmia.

In some cases hyperæsthesia is an early sign of the onset of non-tuberculated leprosy, and will generally be found in the course of the ulnar or sciatic nerves in such cases.

A very interesting fact from a physiological and diagnostic point of view is that disease of the internal organs or viscera often produces areas of hyperæsthesia or tenderness upon the skin, which may in future be used to aid in the localization of the lesions. This subject has been well studied by Head (*Brain*, 1893 and 1894), from whose researches much information may be derived, but the results of which will have to be confirmed in many cases before they can be used as diagnostic guides. (See article on Pain.)

Pain in the skin is very various in its manifestations, and nearly always is due to functional nervous troubles. Duhring has noted a boring sensation in some cases. It should direct the physician's attention to the possibility of hysteria or tabes dorsalis.

Pruritus or intense itching of the skin may be due to contact with some irritant, but its presence, if persistent, particularly if widespread or near the genitals, should always raise a suspicion of diabetes mellitus, or chronic lead-poisoning, or gout, or chronic contracted kidney. Very rarely opium may produce a pruritus, and jaundice is nearly always accompanied by some itching. Pruritus about the anus is often due to piles, gout, and diabetes.

Finally, one important point is to be remembered, viz., we cannot attempt to make a general diagnosis merely from a study of the areas of anæsthesia or other perverted sensibility of the skin in any case. The results obtained from studies of the sensation of the skin are only to be used as additions to the motor and other symptoms which will be found discussed under the chapters on the limbs.

CHAPTER VII.

THE THORAX AND ITS VISCERA.

The inspection of the normal and abnormal chest—Their topography—Alterations in the shape of the thorax—The rhythm of the respirations—The results of using inspection, palpation, percussion, and auscultation in health and disease—The characteristic signs and symptoms of the various diseases of the thoracic organs.

THE chief contents of the thoracic cavity consist of vital organs, which are, unfortunately, only too often subject to disease. A careful study of the signs associated with the normal functions of these parts is, therefore, of importance, as is also that of the symptoms indicating pathological changes. While it is true that in many instances patients present themselves to the physician with well-marked objective and subjective symptoms pointing to abnormalities in the organs of the chest, it is also a fact that in many others none of these signs exist, or they exist in such an indefinite manner that the physician's attention is not attracted to them, and as a result important thoracic changes from the normal are overlooked or made light of. We base our diagnosis of the character of a case on the changes which we find in the surface of the thorax as to its contour and as to its movements, on the respiratory and cardiac sounds, and on the other physical signs about to be described.

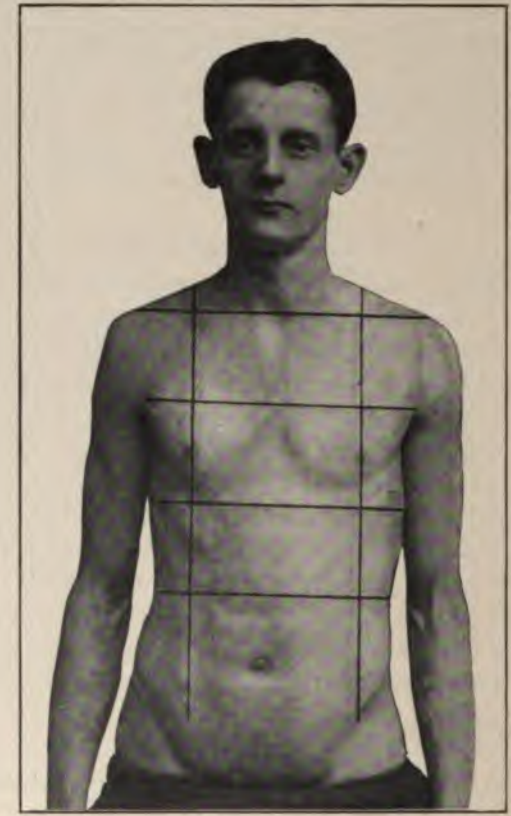
The measures used in the physical diagnosis of the diseases of the thoracic organs are Inspection, Palpation, Percussion, and Auscultation.

Inspection.

Before we proceed to the study of the alterations produced by disease in this portion of the body, we must have a clear conception of the appearance of the chest in health.

Inspection of the normal chest when free from clothing will reveal the fact that it is conical in form, the broader part of the cone being in the upper portion. Above the clavicles there is usually a slight depression (the supraclavicular fossa), and below the clavicles, which may be somewhat prominent, there is a slight convexity which extends as far down as the fourth rib. This convexity varies considerably according to the muscular development of the individual, the formation of the bony portion of the chest-wall, and the deposit of fat in the subcutaneous tissues of the chest.

FIG. 86.



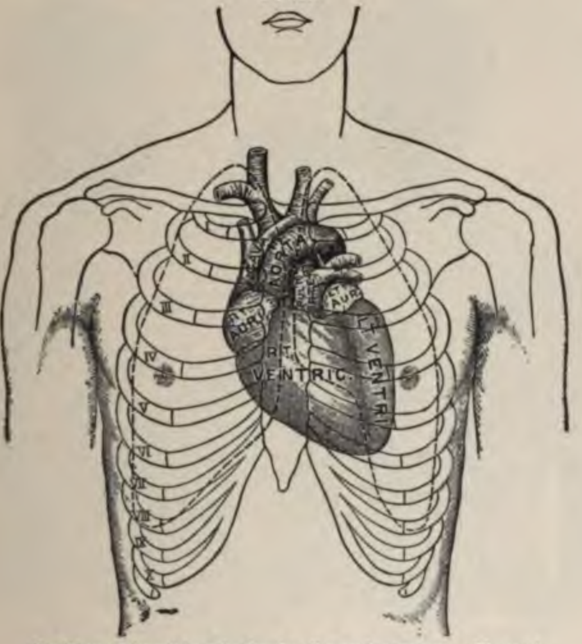
The regions of the anterior aspect of the chest. The vertical lines are called the mammillary lines. The upper squares form the superior thoracic area; lower squares form the inferior thoracic area, and the lowest the epigastric.

The nipple is by no means as definite a landmark as is sometimes thought, as its position, in respect to the ribs under it, varies greatly in different individuals; and it is still further altered in its position by the presence of much fat under it, or, again, in multiparous women owing to the relaxation of the breast. In the average adult male or virgin female the nipple is on a level with the fourth rib or fourth interspace. The ribs in a well-developed person are not prominent in the upper two-thirds of the chest, but in the lower third are more readily seen, particularly at the sides, because of their thin covering by muscles and the subcutaneous tissues and the skin. The sternum in front and the spine behind are normally in the middle line. Over the top of the sternum is a depression called the episternal notch.

The result of lateral examination of the normal chest when compared with the front view will show that the antero-posterior diameter is less than the lateral diameter.

The surface of the chest anteriorly, posteriorly, and laterally, has been arbitrarily divided by imaginary lines into spaces, as shown in the accompanying figure. (Fig. 86.) The lines running fróm the middle of the clavicles downward through the nipple are called the mammillary lines. The parasternal line, not shown in the figure, is a vertical line half-way between the middle of the sternum and the mammillary line; and a line running down the side from the axilla is called the mid-axillary line. These artificial divisions enable us to describe the locality of signs and symptoms.

FIG. 87.

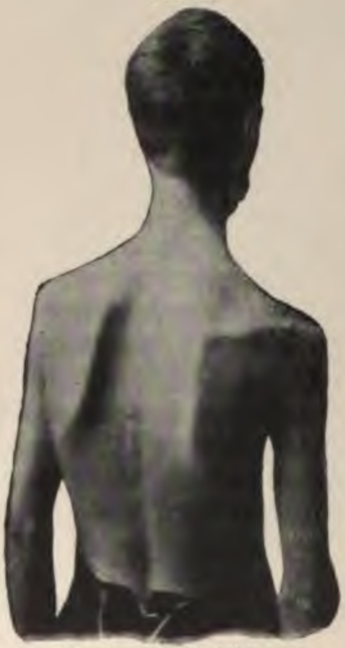


Position of heart in relation to ribs and sternum. (TYSON.)

If we could see through the chest-wall, we would find that the lungs extend above the clavicles. Immediately back of the inner end of the left clavicle is the beginning of the innominate vein, and back of this, again, the common carotid artery. On the right side the innominate artery bifurcates just behind the junction of the sternum and clavicle. The figure just given shows the relation of the cavities of the heart and its great vessels to the chest-wall. (Fig. 87.)

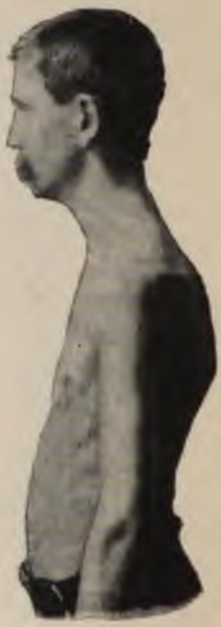
Anteriorly the lung extends downward as far as the sixth rib on the right, but the dome of the liver reaches to the level of the fourth interspace. On the left side the lung extends a little lower than on the right side. Laterally the lung on both sides extends to the ninth rib in the mid-axillary line. Posteriorly on the right side the lung extends as low as the tenth rib, and on the left side as low as the ninth.

FIG. 88.



The alar chest of phthisis.

FIG. 89.



Side view of same patient.

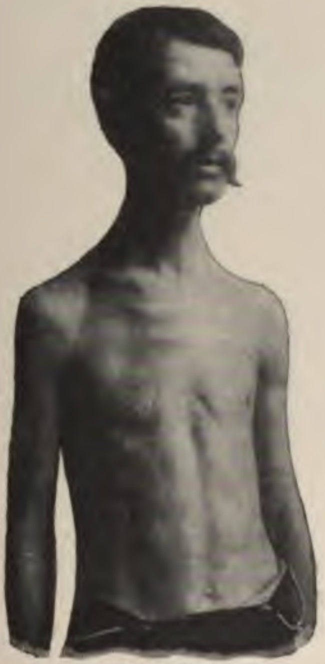
Marked variations in the shape of the chest occur in healthy individuals without possessing any direct pathological significance.

Thus, it is very common to see one shoulder slightly higher than the other, and, in the case of clerks or persons who work much at a desk, the left shoulder is very apt to be somewhat elevated. Occupations which cause the individual to assume certain positions, or to use certain muscles continually, also cause variations in the contour of the thorax.

INSPECTION OF THE ABNORMAL CHEST. The configurations of the chest which show a tendency to disease or the results of attacks of disease are numerous.

The most familiar of these is the so-called phthisical chest, which has been called the "alar chest," because the scapulæ stand out from the back like wings. (Fig. 88.) The antero-posterior diameter, particularly in the upper two-thirds, is very slight, and instead of convexity of the anterior surface there may be flattening or hollowness. (Fig. 89.) This area scarcely moves on inspiration, but the lower third, which is bulging, moves markedly with the respiratory efforts, as does also the epigastrium. The shoulders are very

FIG. 90.

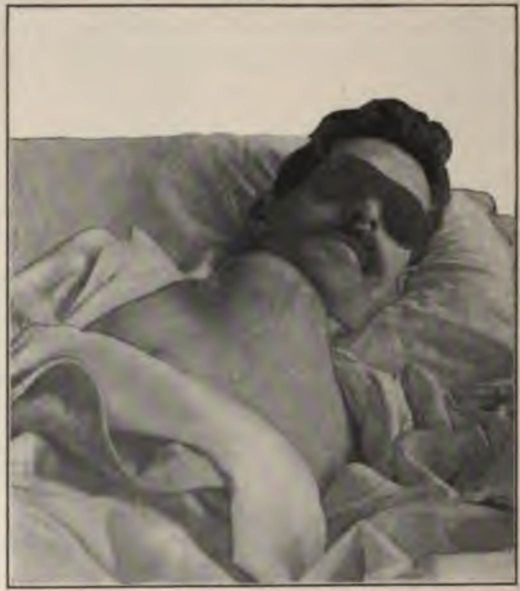


Phthisical chest.

sloping; the neck, anteriorly, recedes at the episternal notch, but springs forward toward the Adam's apple and the chin. The ribs in the phthisical chest fall downward toward the belly from their points of origin, instead of coming forward in a normal curve. (Fig. 90.)

If, on the other hand, the chest bulges anteriorly and posteriorly to such an extent that the antero-posterior diameter is greater than, or equal to, the lateral diameter, and if this bulging is fairly uniform, the shoulders being elevated, the back rounded, and the neck short in appearance from the raised shoulders, the patient is probably a sufferer from emphysema of the lungs. This chest is often called the "barrel-shaped chest." (Fig. 92.) The chest-wall moves very little or not at all with the respiratory movements, which are chiefly diaphragmatic.

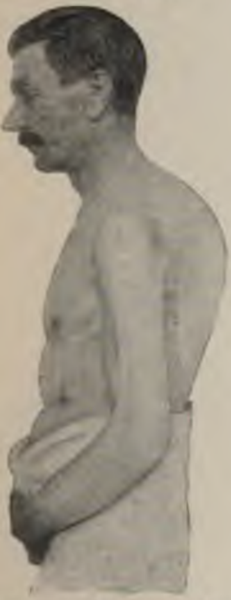
FIG. 91.



Aneurism of ascending arch of aorta.

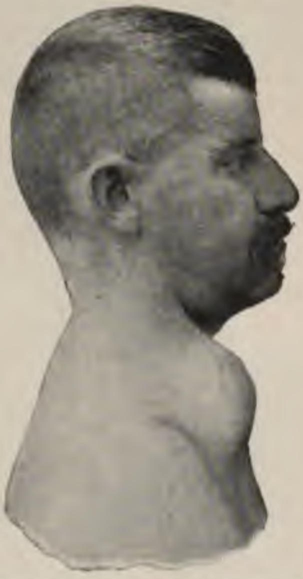
Bulging of the chest-wall results, in its most diffused type, from the presence of chronic pleural effusion and pneumothorax; bulging of a limited area also arises from cardiac hypertrophy, particularly that occurring in childhood; from aortic aneurism, causing bulging by pressure (Figs. 91, 93); from pericardial effusion; and, finally, from mediastinal growths. Marked bulging over the lower part of the chest on the right side should cause us to look for some hepatic affection as well as to examine for pleural effusion, and, if the bulging is low down on the left side, to examine for pleural effusion or enlargement of the spleen.

FIG. 92.



Emphysema of the lungs. Shows barrel-shaped chest.

FIG. 93.

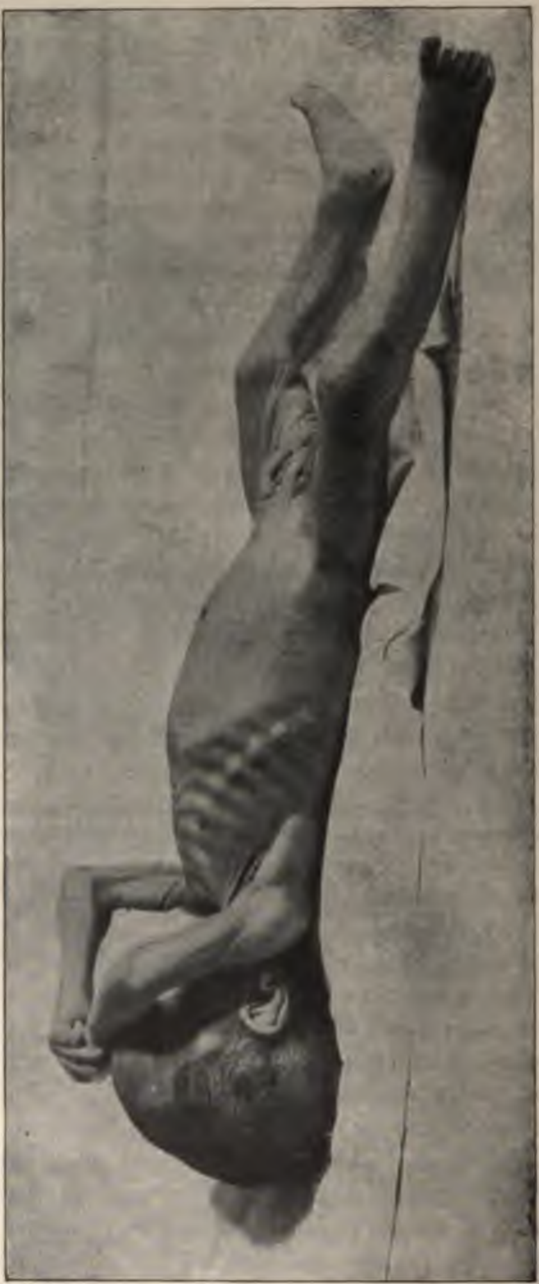


Bulging of the chest-wall, with erosion of ribs, from aortic and innominate aneurism.

Bulging or protrusion of the sternum and the cartilaginous portions of the ribs attached to it is called "pigeon breast," and is due either to rickets or to the presence of some obstruction to respiration of a more or less chronic character during the time the chest-wall was soft and capable of being moulded. Sometimes on each side of the sternum, over the costal cartilages, there is seen a groove or depression as the result of rickets. In other cases a depression or groove extends from the ensiform cartilage backward on either side toward the spine. This is called "Harrison's groove," and is developed in children with poor bony systems, as the result of repeated attacks of asthma or other obstructive respiratory difficulty.

When examining the chests of children the physician will often notice swellings of the tissues at the costo-cartilaginous junctions,

FIG. 94.



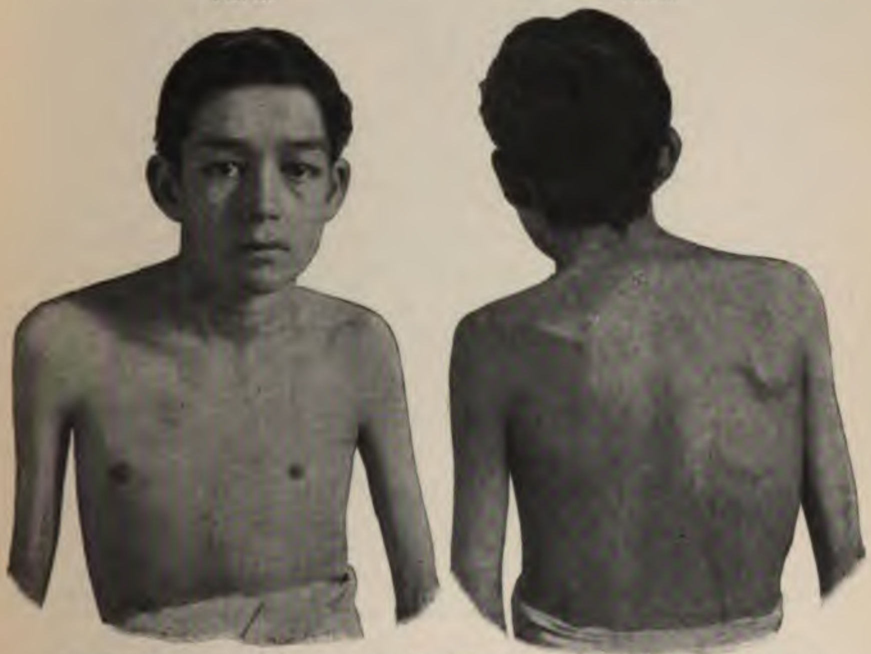
Rhachitic rosary. (BARBOUR.)

which look and feel to the touch like large beads under the skin. These beaded ribs are indicative of rickets, and are a manifestation of the general tendency to epiphyseal enlargement. This beading is usually most marked on the lower ribs. (Fig. 94.)

Finally, unilateral bulging of the chest may be due to curvature of the spine, which part of the body should always be examined before a diagnosis as to deformity of the chest is attempted.

Shrinkage of the chest in one part may be due to the contraction of old pleural adhesions (Figs. 95 and 96). It is sometimes seen over the diseased area in pulmonary tuberculosis, and may be apparently present, but in reality due to wasting of the tissues covering the part.

FIG. 95. FIG. 96.

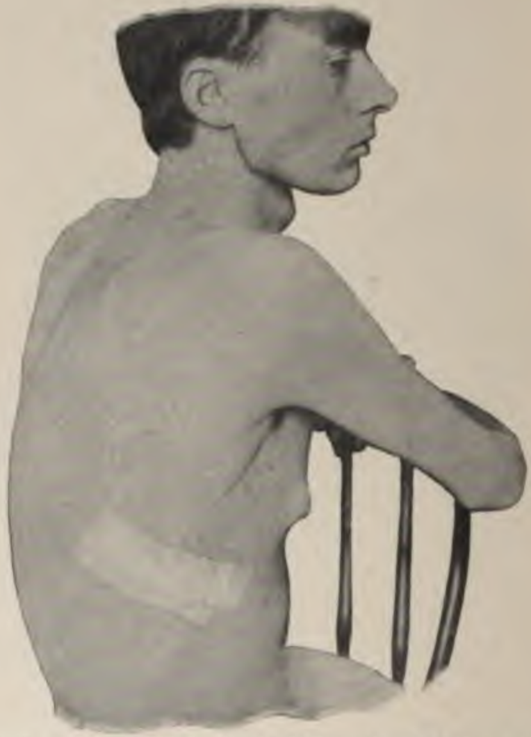


Showing shrinkage and partial collapse of left side of chest and distortion of spinal column due to chronic tubercular pleurisy in a boy of fifteen years. (From the author's wards in the Jefferson Medical College Hospital.)

Marked enlargement of the mammary gland on the affected side is sometimes seen in pulmonary tuberculosis, particularly in males. (Fig. 97.)

The shape and surface of the chest having been studied, we can go further and learn much from its movements in respiration: first, from the rapidity of respiration; second, from the respiratory rhythm; third, from the character of the breathing; and, fourth, from the movements of the ribs.

FIG. 97.



A case of empyema of the right side, showing obliteration of the intercostal spaces and hypertrophy of the mammary gland. (From the author's wards.)

When counting the respirations the physician should always endeavor to do so without letting the patient know what he is doing, since it is difficult for many persons not to control their breathing when their attention is called to it. Generally the eye can detect the frequency of the breathing by simply watching the movement of the chest, or the information can be gained by resting the hand on the abdomen or thorax, while the wrist is also held and the doctor is apparently taking the pulse. In the newly born child in perfect health the respirations are often as high as forty-four, but in the adult male at rest they are usually about fourteen to sixteen per minute. During sleep the number may fall to eight or ten. The ratio of pulse to respiration is usually four to one, but in rare instances in disease it may be one to one.

Rapid respirations not due to any recent sudden exertion are nearly always indicative of respiratory trouble, primary or secondary. If the primary trouble be in the lung, it will probably be due to croupous pneumonia, catarrhal pneumonia, severe bronchitis, asthma, tuberculosis, pulmonary abscess, or tumors of the lungs. If it be due to secondary lesions in the lung, it may rise from pulmonary œdema due to nephritis, from congestion or hypostatic exudation as the result of a weak heart, from pulmonary embolism, from a pleural effusion which seriously interferes with the action of the lung or lungs, from growths in the mediastinum pressing upon bloodvessels and so causing exudation into the lungs or pleura, and from ascites or abdominal growths pressing upon the diaphragm. Usually in these states the respirations will be not only more rapid than normal, but difficult or labored. Sometimes in hysterical rapid breathing the respirations reach 150 per minute. This is not voluntary, and the diaphragm moves very little, the chief breathing being costal. If the lungs be clear of trouble, then the difficulty may be present in the trachea or larynx, either as the result of spasmodic contraction of these passages or because they are occluded by growths, such as papilloma or malignant growth, inside or outside, or aneurism which may act by pressure, thereby narrowing the tube. Any agency which interferes with the proper oxygenation of the blood causes rapid breathing unless at the same time the respiratory centre is depressed.

There are, moreover, several other causes which affect the character of the respiration without affecting the larynx or lung-tissues directly or indirectly. These are fever, which acts as a respiratory stimulant, and excitement, nervous or mental, particularly that of hysterical patients. Again, apoplectic seizures, uræmia, and diabetic coma may be accompanied by rapid breathing.

The respirations are slowed or decreased in number by great obstruction to the entrance of air into the lungs from any cause, so that it is difficult to inhale the air, by the action of poisons made in the body, as the poisons of uræmia and diabetes; by the effect of poisons swallowed or absorbed in other ways, notably opium, chloral, aconite, chloroform, or antimony.

The rhythm or relative time of inspiration, expiration, and the pause is in health in the mouth and trachea as follows: If 10 represents a complete respiratory cycle, inspiration is represented by 5, expiration by 4, and the pause by 1. If it is difficult for air to enter the chest, as in spasmodic croup, the inspiration is much prolonged. This prolongation is also sometimes very marked in cases of paralysis of the posterior crico-arytenoid muscles. If there is difficulty in expelling the air, the expiration is prolonged, as in asthma and in emphysema.

Labored breathing (dyspnœa) is seen in all cases in which the blood cannot be provided with sufficient oxygen owing to obstruction to the entrance of air into the chest, to spasm of the bronchioles, or to the occluding of the air-vesicles by any form of exudate, croupous, catarrhal, or serous. These conditions may be primary or secondary to disease elsewhere, as in uræmia or cardiac disease. Inspection of the chest in such a case shows great activity of the accessory respiratory muscles, such as the sterno-mastoid, the scaleni, the pectorals, and the abdominal recti. The nostrils are dilated and the face is anxious. The posture of the patient is that of sitting up in bed.

Sometimes when the chest is flexible, as is that of a child, the inspiration is jerking when there is obstruction to breathing. This is due to the fact that the chest is forced into expansion by muscular effort, and at the same time is subjected to the external atmospheric pressure, while the air enters the lung slowly and irregularly owing to the obstruction.

The most remarkable change in rhythm is the so-called Cheyne-Stokes breathing, in which the patient after a pause of several seconds begins to breathe with gradually increasing rapidity and depth, and then, after reaching an acme of hurried respirations, gradually decreases their rapidity and depth till they fade to nothing, when, after a pause, the same process is repeated. This breathing is seen commonly in apoplexy, in uræmia, in brain-tumor, in cerebro-spinal fever, in meningeal tuberculosis, in some rare cases of cardiac valvular disease, probably as the result of embolism, and in hæmaturic malarial fever. Rarely it occurs in cases of acute febrile disease, as typhoid fever, scarlet fever, pneumonia, whooping-cough, and puerperal septicæmia. It may also be met with in the course of diabetes. Its presence is an exceedingly bad prognostic sign, but cases of recovery after its onset have been observed, and Murri has reported a case in which Cheyne-Stokes breathing lasted forty days, and Sansom one in which it lasted one hundred and eight days. If the cause be an acute disease, recovery is more common after this symptom than if it be due to some chronic process with an acute exacerbation.

The function of breathing and the movements of the chest are closely associated. In men the respiratory movements chiefly affect the lower ribs and the abdominal walls, owing to the fact that as the diaphragm descends it pushes the abdominal contents downward, so causing abdominal bulging. In women, however, this is not so marked, and the breathing is chiefly costal, the upper part of the chest moving more than the lower (costal breathing). If abdominal breathing is absent in a man and is replaced by breathing of the costal type, we can be assured that the movements of his diaphragm are impaired by the pressure of fluid in the abdomen (ascites); by peritonitis, causing fixation of the diaphragm, owing to pain; by the presence of large growths in the abdomen, or by great enlargement of the liver and spleen. Other possible causes would be a subphrenic abscess or a greatly enlarged cystic kidney, or hydronephrosis.

If the costal breathing of a woman is absent, there is nearly always some pulmonary cause for it, such as faulty development, or, if due to disease, its absence arises most commonly from tuberculosis or pleurisy, or old pleural adhesions which bind down the chest-wall.

It is also necessary to notice the extent of the chest-movements. These are very limited in the characteristic chest of a person having a tendency to tuberculosis, and in the barrel-shaped and rigid chest of emphysema of the lungs. Deficient respiratory movement is not only a predisposing cause of lung disease, but an important diagnostic sign. When one side of the chest moves more than the other to a considerable extent, we suspect, in the side which moves slightly, a pneumonia, a pleuritis, a pleuritic effusion or adhesion, tubercular consolidation or fibroid lung, provided that the patient has not naturally a greater development on one side than the other, or has not pursued a trade or occupation causing unilateral hypertrophy.

While inspecting the surface of the chest the physician should also note the presence or absence of enlarged or pulsating blood-vessels on its surface or about the base of the neck. The cervical vessels are commonly seen to be distended in cases of advanced emphysema of the lungs and in chronic bronchitis. Systolic pulsation of the jugular veins indicates tricuspid regurgitation. Again, in cases of thoracic aneurism pressing upon the superior vena cava and innominate veins we find spongy venous masses above the clavicles, and the veins of the trunk and arms may be engorged. Intrathoracic growths produce similar symptoms.[1] Pulsation in the cervical vessels is also sometimes seen in cases of severe anæmia and in cases of aortic dilatation with regurgitation.

In this connection should be mentioned the "*wavy breathing*" met with most commonly in pneumonia, a condition in which inspiration and expiration do not seem to occur regularly or evenly all over the chest, one part filling or emptying a moment before the other. This usually indicates a grave pulmonary condition.

Sometimes when a patient is placed flat on his back with his feet pointing straight toward a window (cross-lights being excluded) and the chest exposed, the following phenomenon can be observed during forced respiration: along both axillæ a sort of shadow is seen to descend during deep inspiration from about the seventh to about the ninth rib, passing up again during expiration. It is best seen in spare, muscular young persons of either sex. The observer should stand with his back to the light.

This phenomenon is nearly or entirely absent in the following condition: (1) Fluid or air in the pleural cavity. (2) Obliteration of the pleural cavity by adhesions. (3) Advanced emphysema of the lungs. (4) Pneumonias of the lower lobe. (5) Intrathoracic tumors low down in the chest. It is called Litten's sign.

Slight and limited pulsations on the chest-wall elsewhere than over the apex-beat, may be due to many causes.[2] (See Palpation.) When they are seen in the second or third interspace on the right side, they are due as a rule to displacement of the heart, which has been drawn to the right by mediastino-pericarditis or contraction of the right lung and pleura as a result of chronic disease. If the pulsation is lower than this, it is usually due to a dilated right auricle, or displacement of the entire heart, as in a left-sided hydro-, pneumo-, or pyothorax. (See Plate XIV.) If the pulsation be on the left side of the sternum, then it may arise from a displaced apex-beat due to effusion, or to retraction of the pleura which has become adherent to the pericar-

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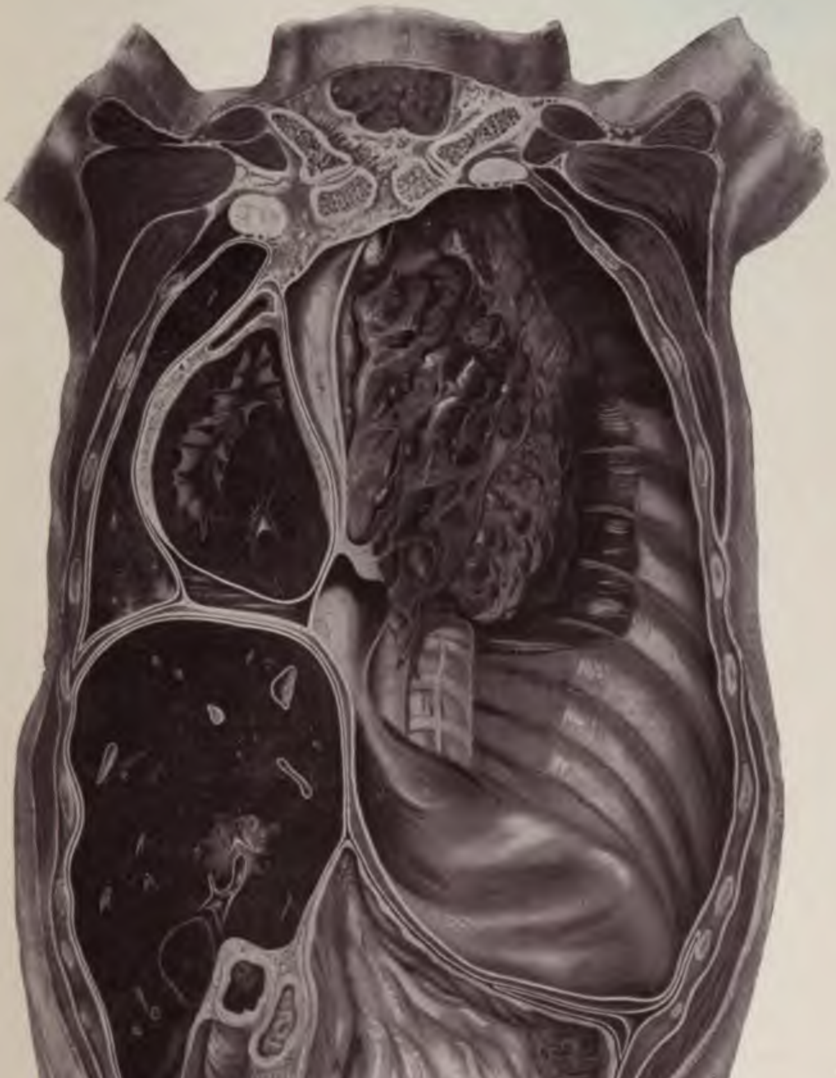
[1] See "The Pathology, Clinical History, and Diagnosis of Diseases of the Mediastinum" by the author. Fothergillian Prize Essay of Medical Society of London for 1888.

[2] These are spoken of here because they can often be seen yet cannot be felt with the finger-tips.

PLATE XIV.



A frozen section made in a plane lying 5 cm. in a postero-lateral direction from the nipples and too near the anterior surface to show the large vessels of the chest and neck. The patient died of acute left-sided Pneumo-thorax, resulting from tuberculosis. The section reveals the collapsed left lung, the air space about it, the heart pushed far to the right in its sac and the anterior border of the right lung compressed by the heart. The right lung is is also greatly displaced, and the air has pushed the diaphragm downward on the left side. (Ponfick.)

dium, or fibrosis of the lung may be the cause. Finally, there may be marked epigastric pulsation. This is due to displacement of the heart by left-sided pleural effusion, which pushes the heart to the right and downward, to hypertrophy of the right ventricle, to pulmonary emphysema, often a cause of enlargement of the right side of the heart, and, finally, it may be due to transmitted pulsation of the abdominal aorta. If the latter is the cause, it can usually be determined by deep palpation that the pulsation arises from this vessel. Rarely it is due to the transmission of the aortic impulse by a tumor which overlies the artery. If this is the case, it will be found that when the patient takes the knee-chest posture the pulsation disappears because the growth falls away from the bloodvessel.

If the epigastric pulsation is in the nature of a systolic retraction, then it is probably due to indurative mediastino-pericarditis.

Rarely, on inspection of the chest anteriorly, a curious retraction of the interspaces near the level of the apex-beat is noticed to occur with each systole of the heart. This is usually indicative of an adherent pericardium, and when on inspection of the posterior surface of the chest such retraction is seen at the level of the eleventh interspace it is called "Broadbent's sign" of adherent pericardium. This movement is supposed to be due to the heart pulling on the central tendon and muscular portion of the diaphragm.

(For the further discussion of the significance and position of cardiac pulsations and thrills, see Palpation below).

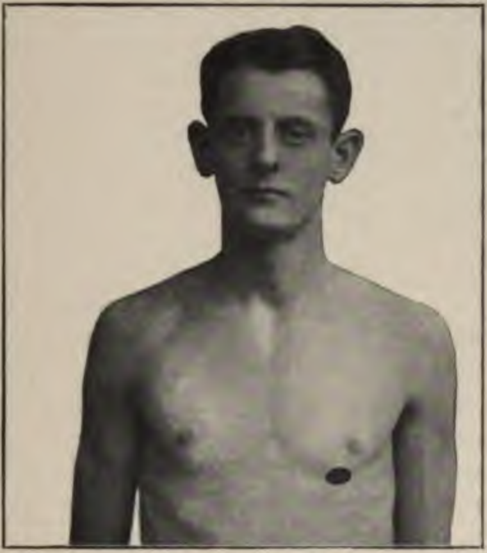
Palpation.

Palpation of the chest is usually performed by placing the finger-tips or the whole hand, palm downward, on the chest. This reveals alterations in its contour and in its elasticity. It will also reveal the ability of the thoracic viscera and the chest-wall to transmit vibrations produced by the voice (vocal fremitus). This so-called vocal fremitus depends upon the fact that below the vocal bands lies a column of air which reaches to the vesicular portions of the lung, and when an individual speaks this column of air is put into vibration, and these vibrations are in turn transmitted to the chest-wall. Of course, a chest-wall greatly thickened by fat or by highly developed muscles will not transmit these vibrations as readily as a thin chest-wall; but aside from these causes of variations in fremitus in health we have a number of causes in disease which greatly modify vocal fremitus. It must be remembered, too, that this vibration is more marked in men than in women and children, because the voice of a man is so much louder and has greater volume. Vocal fremitus is also greater on the right side than on the left, because the principal bronchus supplying this lung is larger than that of the left side, is joined to the trachea at a less acute angle, and is nearer the vertebral column; and, again, as recently emphasized by Cary, the bronchus going to the right upper lobe is given off at a point very near the origin of the right bronchus, and in many cases "fully two and a half inches above the corresponding left bronchial tube." Sometimes this upper tube comes off the trachea directly.

The conditions of disease which cause a decrease in vocal fremitus are pleural effusions of any kind, which not only cut off the transmission of sound, but by their contact prevent vibration of the chest-wall; pneumothorax, which causes collapse of the transmitting medium, the lung; any condition which causes occlusion of a large bronchus, such as tumor or a large mass of mucus, and great pleural thickening. When the vocal fremitus is increased it is an indication of pneumonia, of tubercular thickening or consolidation of the lung, of the presence of a cavity or of tumor in the thorax touching the chest-wall. Fremitus is increased in these conditions because the consolidated lung transmits the vibrations of the air in the bronchial tubes to the chest-wall, or, in the case of a cavity, the sound is transmitted directly to it, and it there causes so great a vibration of the air in the hollow space that the vibration of the chest-wall is marked. (In this connection, see part of this chapter on Auscultation.)

Palpation of the chest-wall will also give information as to the *position* and *character of the cardiac pulsations*. Thus, the apex-beat of the heart in persons standing erect will usually be felt, in those who are not inordinately fat and who are healthy, between the fifth and sixth ribs, about two inches to the left of the sternum. (See Fig. 98.) If the apex-beat is below this level, its depression may be due to enlargement of the heart (hypertrophy or dilatation), to effusion in the pleural cavity on the left side, to pulmonary emphysema causing abnormal descent of the lung and diaphragm, and with it cardiac hypertrophy. Sometimes tumors in the chest produce a similar depression of the apex-beat. On the other hand, if the apex-beat of the heart is felt above the fifth interspace, the heart may be raised by pericardial effusions or adhesions following inflammation, by pleural adhesions or effusions, by abdominal effusion (ascites), by tumors, by distention of the colon with gas, and by great enlargement of the spleen. Displacement of the apex-beat to the left is generally associated with downward displacement, and is commonly due to hypertrophy of the left ventricle, to pleural adhesions, and particularly to pleural effusion on the right side. (Figs. 109, 110.) Displacements to the right are due to hypertrophy and dilatation of the right ventricle, so that the apex-beat is felt in the epigastrium or against the edge of the sternum. Pleural effusion or pneumothorax on the left side may also cause this displacement. (Plate XIV.)

FIG. 98.



Area of normal apex-beat.

The area of the normal apex-beat is about one square inch. In disease this area often extends over several square inches, generally as the result of hypertrophy and dilatation of the ventricles.

The strength of the apex-beat in health depends largely upon the depth of the chest and the thickness of its wall. It is increased in hypertrophy of the heart, when the heart is fully under the influence of stimulants, as, for example, digitalis in full doses, in the early stages of acute fevers, and in great nervous excitement. If hypertrophy is the cause, the apex-beat is usually lower, and more to the left than normal, and the impulse is more or less diffuse. The apex-beat is decreased in force in cases of dilatation of the heart without compensatory hypertrophy, in degeneration of the myocardium, in cases of effusion into the pericardium and in the presence of pulmonary emphysema, which causes the projection of a part of the enlarged lung between the heart and the chest-wall.

Thrills felt in the chest-wall over the heart may be due to abnormalities in the blood-current when valvular disease or aneurism is present. Thus we find thrills in the præcordium, or in the neighborhood of the apex, in disease of the mitral valve, both regurgitant and obstructive, but they are usually much more marked in stenosis than in regurgitation, which latter condition does not cause much palpable thrill, as a rule, except in children. A well-marked thrill at the apex is usually to be considered a sign of mitral stenosis if it is presystolic in time. It is then the so-called "diastolic thrill of mitral stenosis." Thrills in the neighborhood of the second right costal cartilage indicate an aortic lesion, generally that of aortic stenosis, of aortitis, or of aortic aneurism. When thrills are felt in the tricuspid area, namely, in the midsternal region, or a little to the right of it, the lesion is probably tricuspid regurgitation, as tricuspid obstruction is very rare. Sometimes a thrill in this area is due to aneurism of the descending part of the aorta.

Pulsation is felt in the chest-wall in some cases of empyema. In nearly every instance this pulsation, when it occurs, is found on the left side. It is produced by the impulse of the heart against the effusion, and occurs in two forms: the internal, in which the effusion transmits a heaving impulse to the chest; and the external, in which there is a pulsating tumor external the chest-wall. Sometimes this is called "pulsating pleurisy."

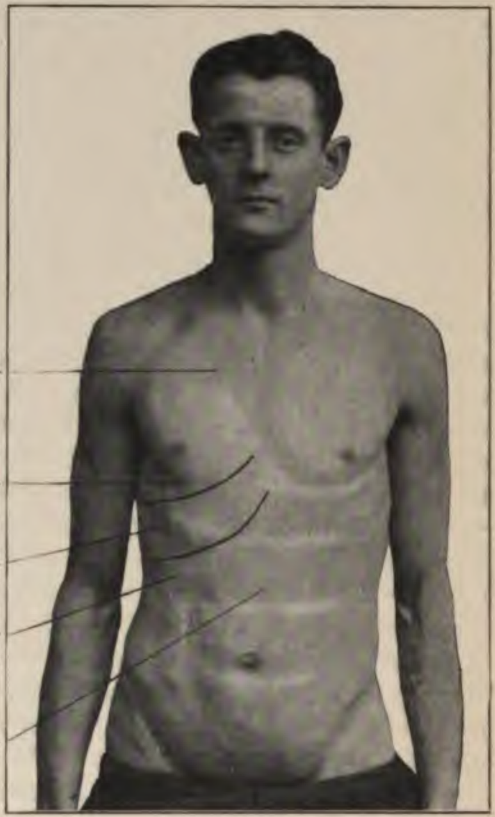
Percussion.

Percussion of the chest is commonly performed by placing one finger, generally the middle one of the left hand, on the chest-wall and tapping it on the back with the tip of the bent finger of the right hand, the movement of the striking hand being entirely a wrist-movement. Sometimes percussion is made by directly striking the chest with the fingers or palm of the hand (direct percussion). Many physicians also employ a percussion-hammer with a rubber head and a pleximeter, or chest-piece, of ivory, celluloid, or glass. Glass is by far the best material for the chest-piece, as it does not produce a note of its own when struck by the hammer, as do the other materials. The disadvantage of this means of percussion is that the physician cannot determine the degree of resistance offered by the surface percussed, which is of the greatest service in many cases of doubtful character, as, for example, in a case in which pneumonia is suspected and the results of the percussion will decide the diagnosis. Care should be taken in performing percussion: first, that similar points on the chest-wall on each side are carefully compared; second, that the finger which is applied to the chest is placed in the same relation to the ribs, or interspaces, on each side when it is struck; and, finally, in studying the effects of percussion the physician should always employ it both during forced inspiration and forced expiration, in order to determine the resonance of the chest with its full quota of air and when it has only residual air.

The resonance produced on percussion is due to three things: first, to the vibrations of the air in the lungs; second, to the vibrations of the chest-wall when it is struck; third, to the vibrations in the pleximeter placed on the chest. The last need be considered as a factor only when a piece of celluloid or ivory takes the place of the finger, for the finger itself does not vibrate enough to alter the note developed. The note produced by vibration of the chest-wall can also be excluded as of little importance unless the chest is very pliable and resilient, as in a thin child, and the blow be delivered very hard. The most important factor in the production of the percussion-note is that first named, viz., the vibration of the air in the chest caused by the blow delivered on the chest-wall. A large part of the percussion-note depends, therefore, upon the amount of air in the chest, the tension of the chest-wall, and the condition of the pulmonary tissues. The sound produced when the healthy chest is percussed is called the normal pulmonary resonance.

In percussion, very different results are obtained by the use of light and heavy blows, and when percussing the chest it is wise, as a rule, to use light percussion, since a heavy blow may produce some resonance in a distant healthy part, and so cause the physician to overlook a small localized area of consolidation, which light percussion might discover. Further than this, it is of great importance that the sense of resistance offered to the finger placed on the chest in percussion be carefully observed, and many experienced men gain more information from this sensation than from the character of the note elicited. In consolidation of the lung the elasticity and resiliency of the chest are impaired, and in pleural effusion they are still more destroyed. The resiliency and elasticity over cavities are marked. The various results produced by heavy and light blows are well shown in the following figure.

FIG. 99.



Light and heavy percussion produce pulmonary resonance alone.

Light percussion develops pulmonary resonance. Heavy percussion reveals dulness due to liver under dome of diaphragm.

Heavy and light percussion gives liver flatness.

Light percussion gives intestinal tympany. Heavy percussion reveals liver dulness due to its lower edge.

Light and heavy percussion reveal intestinal tympany.

The effects of heavy and light percussion in outlining organs.

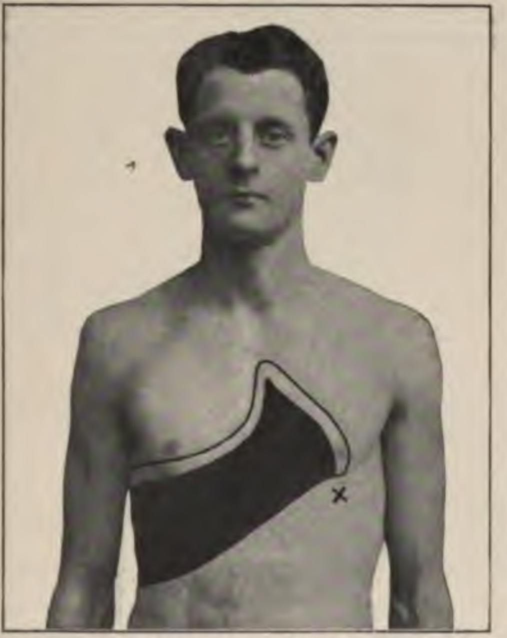
Percussion of the Respiratory Organs. On percussing the right side of the chest anteriorly in the mammillary line we find in health normal pulmonary resonance as low as the fourth interspace or fifth rib, at which point the resonance begins to be impaired, so that at the sixth interspace or seventh rib we find dulness due to the upper border of the liver. The area of partial and absolute hepatic dulness is shown in Fig. 100.

Posteriorly we find on percussion of the right chest that the normal pulmonary resonance begins as high as the suprascapular area, and ends as low as the tenth or eleventh rib. It is much less resonant as compared with the percussion-notes obtained from the anterior aspect of the chest, by reason of the thickness of the chest-wall and the presence of the scapulæ. For this reason pulmonary resonance is best developed posteriorly at the bases of the lungs below the scapulæ. Before percussing the back the patient should be made to lean forward and fold the arms, in order to stretch the tissues and make them tense and as thin as possible.

We can divide the normal sounds produced by percussion into the tympanitic, the dull, and the flat. We can also develop by percussion of the chest in disease what is known as a "cracked-pot sound."

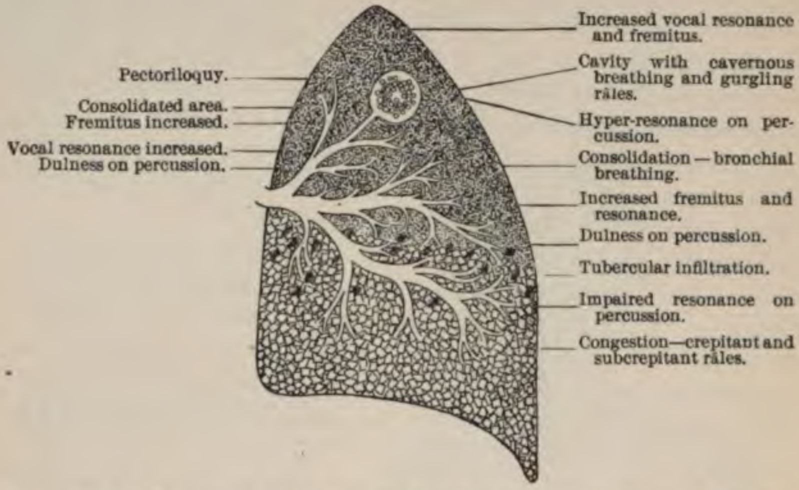
A tympanitic sound is best produced in its most typical form by percussing the epigastrium when the stomach and colon contain some gas. When this sound is produced by percussing the chest it is due to one of several causes, such as a large cavity, pneumothorax, collapse of the lung, or adhesions of the pleural surfaces. A high-

FIG. 100.



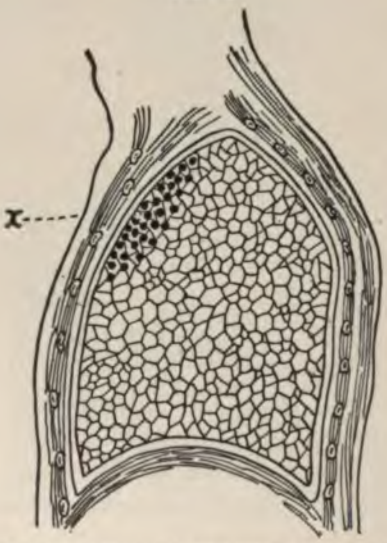
Showing area of partial hepatic dulness (outline) and absolute hepatic dulness (solid), merging into cardiac dulness in enlargement of liver.

FIG. 101.


Pectoriloquy.
Consolidated area.
Fremitus increased.
Vocal resonance increased.
Dulness on percussion.
Increased vocal resonance and fremitus.
Cavity with cavernous breathing and gurgling râles.
Hyper-resonance on percussion.
Consolidation — bronchial breathing.
Increased fremitus and resonance.
Dulness on percussion.
Tubercular infiltration.
Impaired resonance on percussion.
Congestion—crepitant and subcrepitant râles.

Tuberculosis at various stages in one lung, the physical signs depending on the stage. The upper part is consolidated, and contains a cavity communicating with a bronchus.

FIG. 102.



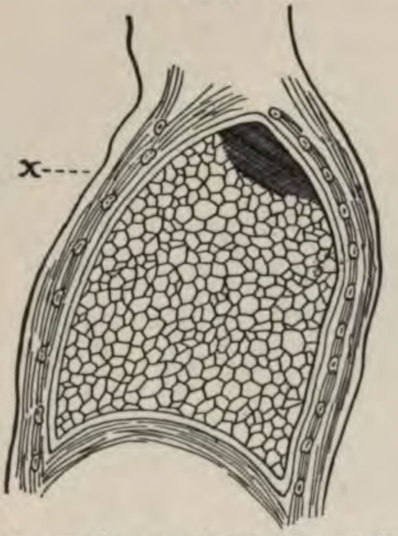
Moderate dulness on percussion at *x* over tubercular infiltration.

pitched note on percussion is also a valuable sign in localizing a deep-seated consolidation, for while consolidation produces dulness on percussion when it is near the chest-wall, it is a common thing for hyperresonance to be found in the chest above the area which is consolidated if healthy lung supervenes.

If the cavity be in the lung itself, it must be of some size and be near the surface to produce a tympanitic note, and, if it communicates with a bronchus, the character of the note will change when the mouth is closed or opened. (Fig. 101.) If the case be one of pneumothorax, with fluid in the chest, changes in the posture of the patient will greatly alter the character of the note.

Consolidation of the lung, as in pneumonia and tuberculosis, as just stated, generally gives a dull rather than a tympanitic note; but if the consolidated area surrounds a very superficially placed bronchus, the percussion-stroke may produce vibration in the air in this tube, and this will cause a note, high pitched in character, which varies as the mouth is closed or opened (Figs. 102 and 103). Collapse of the lung causes a tympanitic note, because the comparatively little air in the lung vibrates as a whole, its vibrations not being stopped as in health by the tense septa and vesicular walls. It is best elicited in cases of pleural effusion over the apex of the chest, into which the collapsed lung has been pushed by the effusion. This is sometimes called "Skodaic resonance." If the

Fig. 103.

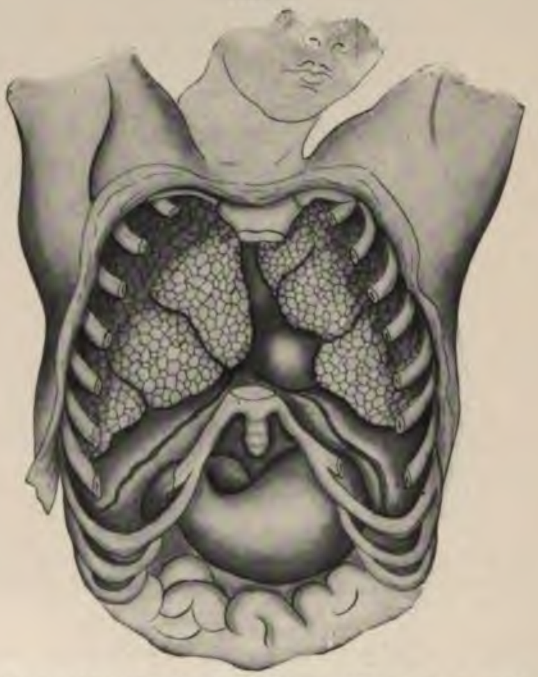


Showing high-pitch percussion-note anteriorly from consolidation posteriorly. The shaded area is the consolidated part; *x* indicates the position anteriorly where the percussion-sound is raised in pitch.

compression is sufficient to consolidate the lung, the tympanitic note is lost. This note is not altered by opening and closing the mouth.

The "cracked-pot sound" is produced in an adult by the sudden expulsion of the air from a cavity through a small opening by the force of the percussion-stroke. It occurs on percussing a healthy child when its mouth is open, the air being forced by the blow from the lung through the glottis. In adults who have a diseased lung the cracked-pot sound most commonly results from the presence of a cavity. It may also be heard in cases of pneumothorax with a fistulous tract opening externally or into a bronchus, in a few cases of pleural effusion in thin-chested persons, and, in rare instances, before consolidation has occurred in pneumonia.

FIG. 104.

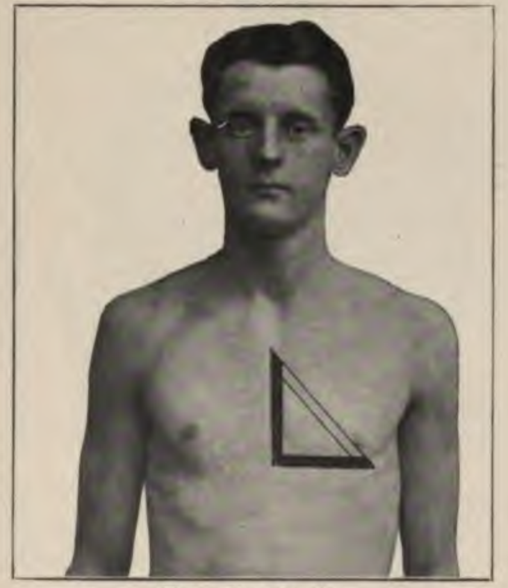


Position of heart partly covered by lungs. (Modified from AITKEN.)

In cases of pleural effusion a flat note on percussion is produced over the effusion, and it is of very much the same character as the sound elicited by percussion of the solid tissues of the thigh.

Percussion of the Heart and Great Vessels. On percussing the chest anteriorly on the left side at the fourth interspace, it will be found that the resonance is decreased by the presence of the heart. At the apex of the chest on this side percussion develops normal resonance, but as we descend in the line situated half-way between the mammary line and the mid-sternal line we find an impairment of resonance at the third rib which becomes in the next inch of descent a very marked dulness, which is produced by the presence of a solid organ, the heart. The impairment of resonance is not complete at the upper border of the heart, because of the fact that the edge of the lung intervenes between the heart and the chest-wall, and so the note which results on percussion is neither the normal resonance of the lung nor the dulness produced by the presence of the heart. (Fig. 104.) The outlines of the normal cardiac dulness on percussion are shown by the following diagram, and they form what have been called the "cardiac triangles." (Fig. 105.)

FIG. 105.

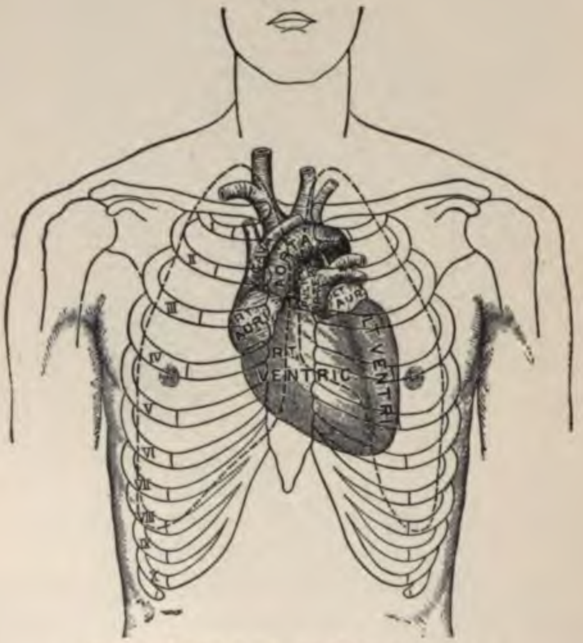


The cardiac triangles.

The large triangle begins at the level of the second left costal cartilage, and extends down the midsternal line to the level of the sixth costal cartilage. The base then extends to the apex-beat, normally situated in the fifth interspace just inside of the clavicular line. The hypothenuse of the triangle joints these points. In this area we have included the partial and total cardiac dulness. The small cardiac triangle, which includes the absolute cardiac dulness, begins at the third costal cartilage and extends to the sixth. The base-line extends to within one and one-half inches of the nipple, and the hypothenuse joins this point with the third costal cartilage at the midsternal line. (Fig. 105.) The borders of the heart really extend further than this, but are not near the chest-wall and are partly covered by lung-tissue as already stated. (See Fig. 104.)

The greater part of the cardiac dulness on percussion is due in health to the presence of the right ventricle, which is nearest the chest-wall. The right auricle also is well forward, while the left ventricle only fringes the edge of dulness to the left. This is well shown in the accompanying diagram. (Fig. 106.)

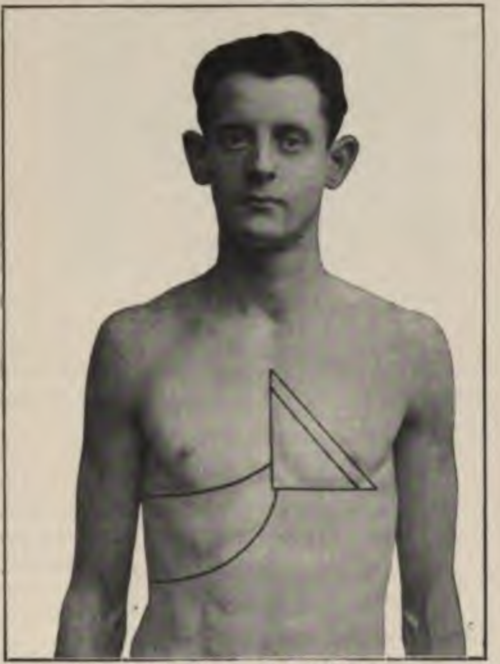
FIG. 106.



Position of heart in relation to ribs and sternum.

When hypertrophy or dilatation of the heart occurs, it will be found that the area of cardiac dulness extends to the right of the sternal line and to the left of the long side of the triangle, while the apex-beat is apt to be displaced downward and to the left. (See Fig. 111.) Great distortion of the triangles occurs as the result of pericardial effusion (Fig. 108), but in this case the heart-sounds will be distant on auscultation and the apex-beat very feeble or lost, whereas in hypertrophy they are exaggerated and the apex-beat forcible. The diagnosis of pericarditis, after the stage of dryness and friction-sound has passed by, is by no means as easily made as some of the text-books would make it appear. One of the most reliable signs of pericardial effusion is that of Rotch, namely, that any considerable dulness in the fifth right intercostal space near the sternum means pericardial effusion, provided pulmonary consolidation and pleural effusions or adhesions are excluded. The writer has, however, often seen this sign present in marked cardiac dilatation; but if, as Ewart has pointed out, there is obliteration of the normal acute angle between the right border of the cardiac dulness and the line of liver dulness, this sign is of value. (Fig. 107.)

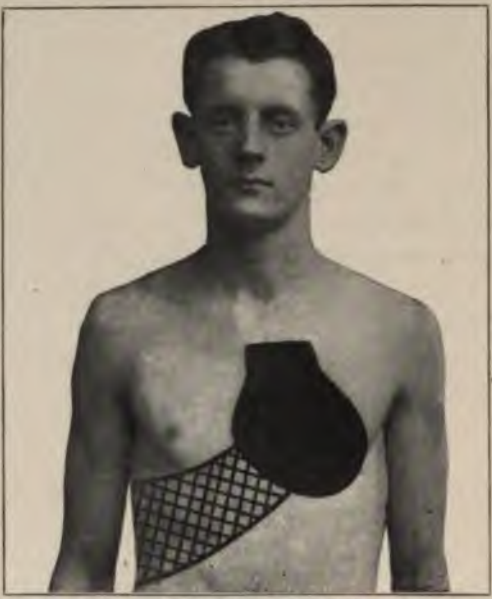
FIG. 107.



Areas of cardiac and hepatic dulness.

In dilatation of the heart the area of the apex-beat is usually diffuse, and the heart-sounds, while feeble, are clearly heard.

FIG. 108.



Outline of percussion-dulness in extensive pericardial effusion. The light shading is the liver dulness.

In this connection the following summary, prepared by Sansom, of the differential diagnosis between dulness due to pericarditis and that due to dilatation of the heart, is of interest:

	Pericarditis with Effusion.	*Dilatation of the Heart.*
Outline of dulness	Dulness pear-shaped, and enlargement chiefly upward.	Dulness not pear-shaped, and enlargement chiefly downward.
Rate of development of dulness	Often rapid, and then characteristic.	Usually very slow, though a rapid dilatation of the heart sometimes occurs.
Impulse and apex-beat	The impulse when present is in the third or fourth interspace; apex-beat tilted upward and outward, or effaced.	Impulse can usually be felt to the left of the lower end of the sternum or in the epigastrium.
Relation of dulness to left apex-beat	Dulness may extend to the left of the apex-beat.	Dulness does not extend to the left of the left apex-beat.
Pain over præcordia and tenderness in the epigastrium	Often present.	Usually absent.
Pulsation in the veins of the neck.	May be present if endocarditis complicates.	Often present when right heart dilated.
Etiology	Usually acute, in course of acute rheumatism, cirrhotic Bright's disease, etc.	Usually chronic; often associated with chronic valvular lesions, fatty and fibroid degeneration.
Fever	Often present.	Absent unless from some complication.

The same author also tabulates the facts in the differential diagnosis between increased dulness due to pericarditis and hypertrophy of the heart as follows:

	Pericarditis with Effusion.	*Hypertrophy.*
Rate of development	Usually rapid.	Usually slow.
Impulse; apex-beat.	Impulse, when present, is in the third or fourth left interspace, and is feeble; apex tilted upward and outward, or beat effaced.	Impulse powerful; if left ventricle hypertrophied, apex displaced downward and outward; if right ventricle hypertrophied, apex displaced downward and inward beat may be in the epigastrium.
Pulse	Weak and quick; may be irregular.	Character of the pulse depends on the side of the heart which is hypertrophied and the cause of the hypertrophy. When left ventricle hypertrophied and no aortic obstruction or mitral regurgitation, the pulse is large and powerful.

In emphysema of the lungs the cardiac triangles may be obliterated by the extension of the lung between the chest-wall and heart.

FIG. 109.

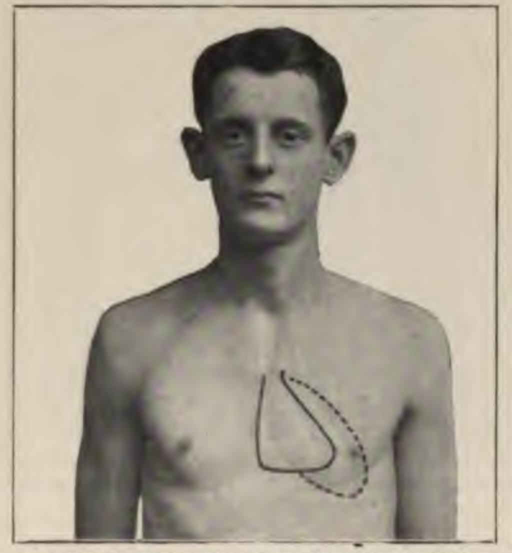
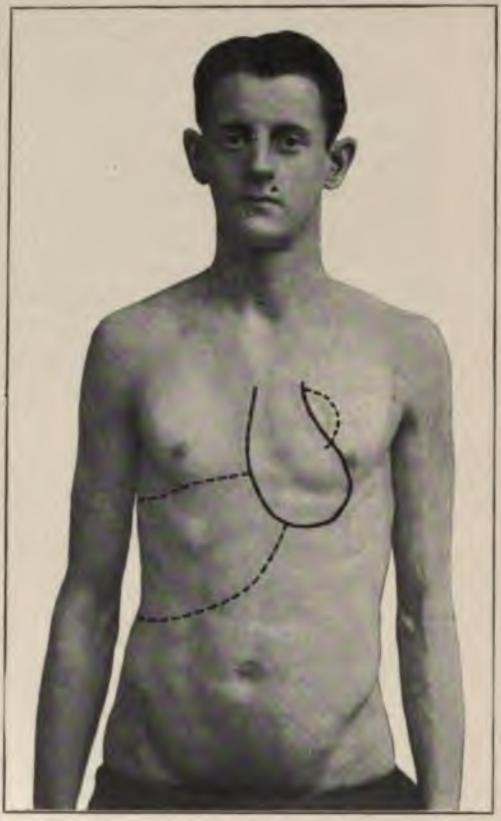


Diagram of the area of the heart in aortic obstruction and regurgitation. The dotted lines show enlargement of the left cavities, especially the ventricle, while the solid lines show the normal area.

They may also be distorted by reason of pleural effusions pressing the heart upward and to the right, or in the case of right-sided pleural effusion the heart may be pushed unduly to the left. Pneumothorax may cause similar results, or, again, old pleural adhesions and conditions may so displace the lungs or heart that the triangles cannot be found.

The various valvular and other lesions of the heart result in alterations in the size of the various cavities without the entire viscus being equally affected. Thus aortic regurgitation causes enormous enlargement of the left ventricle (dilatation and hypertrophy), and aortic stenosis also causes the same enlargement, as a

FIG. 110.



Area of the heart in mitral stenosis. The dotted line indicates the enlargement of the left auricle and enlarged liver. The solid line shows enlargement of the right cavities.

rule, in less degree. Mitral regurgitation causes hypertrophy and dilatation of the left ventricle and some enlargement of the left auricle, and the left auricle is also enlarged in mitral stenosis. Tricuspid regurgitation causes hypertrophy and dilatation of the right auricle and hypertrophy of the right ventricle, and mitral stenosis often has a similar influence over the right side of the heart by damming back the blood into the lungs and right side of the heart. The following diagrams illustrate the deformity of the area of cardiac dulness under these various conditions. (See Figs. 109 and 110.)

Auscultation.

Auscultation of the normal chest reveals in health two sets of sounds: the respiratory and the cardiac.

Auscultation of the Respiratory Apparatus. The respiratory sounds occur in two varieties, namely, vesicular breathing and bronchial breathing. The vesicular sound is heard in its most typical form over the apices of the lungs anteriorly, the latter at the inferior angles of the scapulæ posteriorly. We may listen to these sounds by placing the ear directly against the chest, or by the use of a single or a binaural stethoscope, but neither of these instruments is satisfactory for this purpose. The patient must be in an unconstrained position, as should be that of the physician, and if the ear is placed against the chest, or a single stethoscope is used, the face of the physician should always be turned away from that of the patient, because the breath of a sick person is often very disagreeable and the breath of the doctor may be equally annoying to the patient. Care should be taken in the use of the stethoscope to see that the edge of the bell in its entire circumference is in close contact with the chest-wall.

The respiratory sounds consist, as already stated, in the vesicular murmur and the bronchial or blowing sounds, which are sometimes designated by the term tubular breathing. In the vesicles the air is subdivided into many minute parts, whereas in the bronchial tubes it moves along in a column. Whatever may be the actual cause of the production of normal vesicular breathing, we know that when it is present it signifies a healthy pulmonary parenchyma, and when absent one more or less diseased.

Bronchial breathing, normal in the bronchial tubes, becomes an abnormal sign when it is heard in an area in which vesicular breathing should be present, as will be shown shortly.

After determining the fact that the sounds of normal vesicular breathing are present in the anterior parts of the chest, or that those of bronchial breathing can be heard between the shoulders, we next take note as to the relative duration of the inspiratory and expiratory sounds. Normally in the perfectly healthy chest the ratio of the expiratory sound to the inspiratory sound is as one to three, although if the volume of air itself be measured the duration of expiration is six to five. In other words, so far as auscultation of the vesicular portion of the lung is concerned, inspiration is far longer than expiration. Just at this point we learn one of the most important points in the physical examination of the chest, namely, that while the expiratory sound may be entirely absent in health, any marked increase in its length and loudness, so that it equals or exceeds the inspiratory sound, is a sign indicative of some diseased state which impairs the elasticity of the lung, such as early tuberculosis, pneumonia, and emphysema.

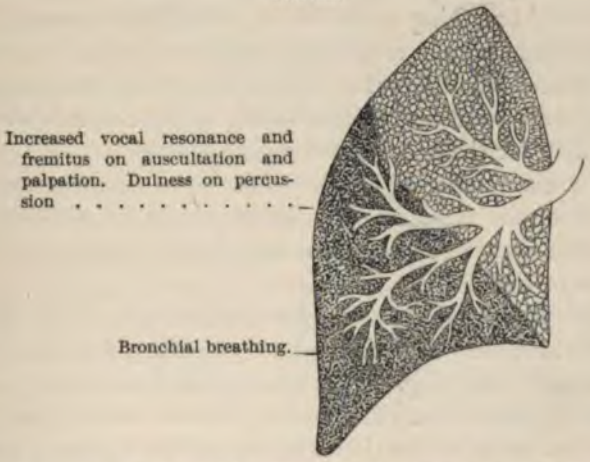
The other variations in the vesicular respiratory sounds differing from those of health are harsh, or, as it is sometimes called, puerile breathing, and irregular breathing. In children, as the term "puerile breathing" indicates, the normal vesicular breathing is loud, clear, and harsh, because of the great elasticity of the lung and the thinness of the chest-wall. If it is exaggerated in a child or present in the area of normal vesicular breathing in adults, it usually indicates some irritation of the bronchial mucous membrane. If it is found in the apices of the lungs in a marked degree, and expiration is prolonged, it is an important and fairly sure sign of early pulmonary tuberculosis.

Sometimes physicians speak of "broncho-vesicular breathing," meaning a breath-sound consisting of both bronchial and vesicular sounds. It is sometimes heard in a healthy person when he breathes superficially, and in disease usually indicates the early stages of pneumonia or early tuberculosis of the lungs. It is of value as a diagnostic sign only when localized in one part of the lung. This harsh breathing of exudation and thickening differs from normal puerile breathing in this important particular, namely, that in the latter expiration holds its normal ratio to inspiration, whereas in disease it is greatly prolonged.

Irregular, "cog-wheel," breathing occurs in the chest of a healthy sobbing child and in that of an hysterical woman, but it possesses pathological significance if it occurs when a full breath is taken, and it is often present as an early sign of incipient pulmonary phthisis.

Bronchial breathing in health is best heard in the posterior part of the chest, as already stated, between the scapulæ and the seventh cervical to the fourth dorsal vertebra. When this bronchial or tubular breathing is heard in other parts of the chest it is a sign of disease, for while the bronchial tubes are distributed to all parts of the lung, the breath-sound which is in them is masked by the sounds of vesicular breathing and muffled by the lung-tissue surrounding them. If this vesicular tissue becomes consolidated by disease, the vesicular murmur is lost and the solid lung transmits the bronchial sounds directly to the ear of the examiner. Bronchial or tubular breathing, or, as it is sometimes called, "blowing breathing," heard in the part of the lung in which vesicular breathing is normally heard, is, therefore, a sign of tubercular or pneumonic consolidation (Fig. 111) or of compression or collapse of the lung above a pleural effusion. Bronchial breathing is also heard in that area of the chest in which vesicular sounds normally predominate, in cases of cavity of the lung, because in such a lung the bronchial sound is transmitted directly to the cavity, and thence to the ear without

FIG. 111.


Increased vocal resonance and fremitus on auscultation and palpation. Dulness on percussion
Bronchial breathing.

Pneumonia of the lower lobe with the physical signs of consolidation.

being impaired by the intervention of healthy lung-tissue. In other words, consolidated tissue and cavities transmit sound better than the normal vesicular portion of the lung, which is a combination of air and vesicular wall. If the cavity be large, we have a loud sound developed by the transmission of the bronchial sound into its open space and by the passage of air through it. This is called cavernous breathing. If the cavity is not very large, or is peculiarly situated in relation to the supplying bronchus, we have what is called "amphoric breathing"—that is, a sound like that produced by blowing over the mouth of an empty bottle. This sound is also rarely heard in cases of pneumothorax in which the bronchial tubes, running near to the pleural cavity, transmit their sound to the air in the pleural space.

It is never to be forgotten that in examining the chest the two sides must be compared, since the well side often gives a standard for that affected by disease, and in doing so it must be remembered that disease not only modifies the signs in the lung in which the morbid process is situated, but also changes the normal signs. Thus pneumonia or pleurisy or pleural effusion causes a louder vesicular and bronchial breathing on the healthy side than is normal, because this lung has to take in more air to make up for the loss of activity on the diseased side. Great care should, therefore, be exercised that the loud harsh breathing of the healthy part in such a condition is not mistaken for the harsh breathing of disease.

There are a number of other sounds heard in the chest in cases of disease of the air-passages. These consist in râles of various kinds, voice-sounds (vocal resonance), friction-sounds, and succussion notes or sounds.

Râles are divided into two chief classes, moist and dry. The moist are subdivided into the crepitant, or crackling, the fine bubbling, and the coarse bubbling. The dry are called sonorous, or sibilant and hissing. Sometimes the sonorous râles are called rhonchi. The fine crepitant râle is best imitated by moistening the thumb and finger-tip, pressing them tightly together and then separating them while they are held near the ear. Another method of imitating the fine crepitant râle is to roll the hair where it grows above the ear between the thumb and fingers; and still another imitation is the crackling of salt when thrown on a fire. This râle is due to the separation of the vesicular walls, which have become adherent because of exudate. It occurs, of course, during the latter part of inspiration, and is an important sign of croupous pneumonia in its early stages before consolidation has occurred. It also is heard in cases of pulmonary collapse and œdema, but not always in any of these diseased conditions. Care should be taken that the fine râles sometimes heard in the chest at the bases, posteriorly, in a person who has been long in one position in bed, are not thought to be indicative of pneumonia, as they are due only to the congestion or accumulation of secretion due to the dorsal decubitus.

Fine bubbling râles occur chiefly in the smaller bronchioles and the coarse bubbling râles in the larger bronchioles, and they are caused by the passage of air through liquid or mucus. These are commonly heard in bronchitis and in pulmonary œdema in the lower parts of the chest, chiefly posteriorly. If such râles are heard anteriorly or in the area for vesicular breathing, they indicate the stage of resolution of a pneumonia, or if this disease has not been present, or is long gone by, they possess the serious import of breaking down of tissue from tuberculosis in the lung. Sometimes these râles are limited to inspiration or expiration. In convalescence from an attack of asthma they occur with a to-and-fro character, and are often musical or tinkling.

If a cavity has formed and liquid is in it, we may hear in the chest a peculiar hollow tinkling, called by Laennec "metallic tinkling." These sounds are sometimes heard over the stomach when this viscus is in motion and contains a little liquid and air.

There is another condition in which metallic tinkling is heard very clearly, and that is hydro-pneumothorax. In this condition there is a continual dropping of liquid from the apex of the chest, or, more correctly, from the compressed lung in the apex of the chest, and as the drops fall through the air in the chest they strike the surface of the watery effusion with a tinkling sound. (Fig. 113.)

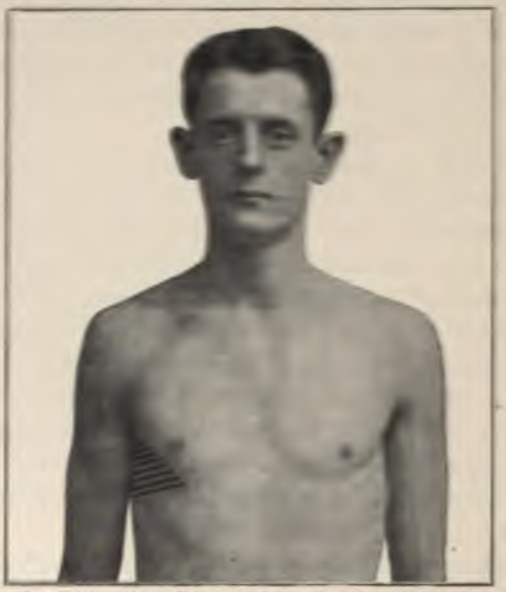
Râles are often removed or altered in character, if not crepitant, by coughing.

It has already been pointed out that dry râles may be divided into the coarse and sonorous and the small or fine sibilant râles. They are produced by the passage of the air, in the large or smaller bronchial tubes, through partly inspissated and sticky mucus. If they are sonorous, the larger tubes are the part involved; if sibilant, the small bronchioles are affected.

It should not be forgotten that harsh breath-sounds made in the mouth or in the nose may cause the transmission of rough sounds or râles into the lungs, which will mislead the physician in his diagnosis if he thinks they arise in the pulmonary tissues.

Friction-sounds in the chest depend upon disease of the pleura or of the pericardium, generally the former. Normally the visceral and parietal layers of the serous membranes glide over one another noiselessly, but when they become roughened by disease a sound of friction is developed. The friction-sound is sometimes so slight as to be almost inaudible, and again so harsh as to sound like a loud creaking, which not only can be heard, but will convey a sensation to the hand when it is placed on the chest. As a rule, friction-sounds due to pleuritis are best heard toward the close of inspiration, and occur only in the early stages of the disease, ceasing with the development of the effusion and perhaps reappearing as the effusion is absorbed. The place where the sound is most audible is near the axilla. (Fig. 112.) An indistinct pleural friction-sound may be emphasized, as pointed out by Abrams, if the patient lies on the affected side a few minutes, and then quickly sits up and stops breathing. The physician places his ear to the chest-wall, and directs the patient to take a long breath, when the sound is developed. If

FIG. 112.



Area in which a pleural friction-sound is usually heard clearly.

a friction-sound is heard at the apex of the chest, tuberculosis will often be the cause of its existence in this locality. Care should always be taken that fine râles are not mistaken for friction-sounds. They can be separated one from the other by the recollection of the facts that râles are modified by coughing, are not affected by deep pressure on the chest-wall, and are usually well diffused, while the friction-sound is not modified by coughing, is intensified by pressure on the chest-wall, and is usually limited to a narrow area.

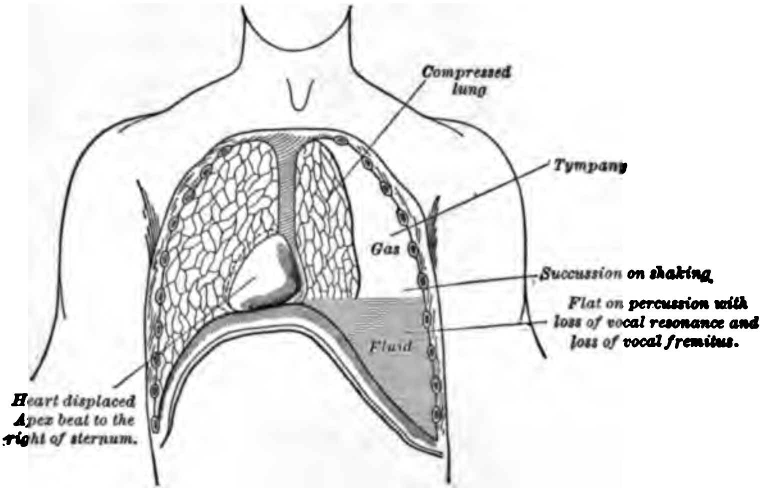
An excellent imitation of a friction-sound is produced by laying one hand over the ear, and then stroking the back of this hand by the fingers of the other hand.

Vocal resonance is closely allied to the sensation called vocal fremitus which is felt on palpation, as already described in this chapter. It is due to the transmission of the voice-sounds down the trachea into the bronchial tubes and bronchioles, and thence through the various portions of the lungs. If a stethoscope is placed in the episternal notch while the patient speaks, and the ear of the examiner which is not closed by the instrument is closed by the pressure of his finger, the voice of the patient will be very clearly heard. If the stethoscope be placed between the vetebral column and the scapula posteriorly—in other words, over the bronchial tubes—the voice also will be clearly heard, but not as clearly as over the trachea, for two reasons: first, because the sound has already been divided into the different bronchial tubes, and, second, because the thickness of the chest-wall muffles it. If the stethoscope be placed over the anterior part of the chest toward the sides in the area of typical vesicular breathing, the sound of the voice will be still more modified, because the sound, like the air that conveys it, is now minutely subdivided, and the vibrations are decreased by the multitude of vesicular walls. Of course, the degree of transmission of vocal resonance is governed largely by the character of the voice, and for this reason it is more distinct in men than in women.

If the patient being examined is a man and has a well-developed voice, it is usually best to have him speak in a whisper, because the full volume of his voice is so great that it will be heard all over the chest, and the nice differences between the transmission of the sound in the healthy lung and in the diseased area cannot be distinguished. Usually we get the patient to speak by asking him to repeat his name or to count "one, two, three." The unemployed ear of the physician should always be closed, and the counting or speaking should be continued only while the physician is actually listening to the chest.

In diseased states of the lung we find the resonance is increased by those changes which aid in the transmission of the sound and decreased by those changes which obstruct its transmission. As pointed out when speaking of vocal fremitus, a solidified lung and the opposite state, namely, a cavity, transmit sound better than healthy tissue, which is partly air and partly lung-tissue. We find, therefore, that the vocal resonance, or the sound of the voice of the patient when he speaks, is increased in pneumonia, in tubercular consolidation, and in cavity; and is decreased in cases of emphysema, or in cases in which a pleural effusion separates the lung from the chest and deadens sound. Vocal resonance, however, may be increased over pleural effusions, particularly the resonance of the whispered voice. This is called "Baccelli's sign," and Baccelli claims that it serves to separate serous effusions from purulent effusions, because in his experience it is absent in the latter class of cases and present in the former.[1]

FIG. 113.


Compressed lung
Tympany
Gas
Succussion on shaking
Flat on percussion with loss of vocal resonance and loss of vocal fremitus.
Fluid
Heart displaced Apex beat to the right of sternum.

Left-pyothorax. (After Maydl.)
Metallic tinkling may be produced by the fluid dropping from the apex of the chest into fluid below.

When a cavity is situated near the surface of the lung so that the sound of the voice is transmitted to it and from it through the chest-wall with unusual clearness, the sound so clearly heard is called "pectoriloquy." It is usually very marked over a cavity connected with a bronchial tube.

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[1] This sign is mentioned here for what it is worth. The writer has never been able to use it with success.

Sometimes when the voice sounds through the chest-wall as if it were of a bleating character it is called "ægophony." It is usually heard at the angle of the scapula, near the margin of a pleural effusion, and is supposed to be caused by compression and partial occlusion of a bronchus.

Finally, in pyo- or hydro-pneumothorax, if the ear be placed against the chest and the patient is shaken, we have developed a splashing or slopping sound, called "Hippocratic succussion." It is not always heard in these cases, and may be developed when a large cavity in the lung is partly filled with liquid. (Fig. 113.)

The healthy physical signs, and the variations from the normal signs met with in diseased conditions of the lungs, have now been discussed. The next step is to group these various signs with other characteristic symptoms in order that we may obtain a complete picture in the diagnosis of a given disease.

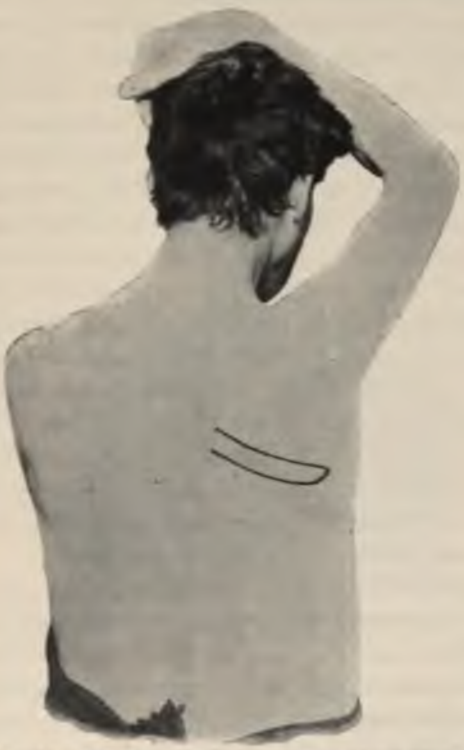
The Diagnosis of Respiratory Affections.

CROUPOUS PNEUMONIA. Let us suppose that a patient, previously in health or without any serious pulmonary complaint, is found, after a physical examination of his chest, to have rapid breathing, a somewhat anxious expression, a bright eye, and a dusky flush on one or both cheeks. Palpation discovers a hot, fevered skin, which is dry or more rarely moist, and increased vocal fremitus over both sides of the chest, more marked on one side than the other. Percussion reveals impairment of resonance over the area where fremitus was found most increased, and auscultation in this area shows bronchial breathing, fine crepitant râles, and increased vocal resonance. Under these circumstances we have before us the physical signs of *acute croupous pneumonia*. The pulse is apt to be rapid, but not so fast as the respiration would lead us to suspect, for it is a characteristic of this disease that the respirations are out of proportion to the pulse. The diagnosis is confirmed by the presence of pain in the side affected, by the cough, the rusty, sticky sputum, and the history that the illness was sudden in onset and was initiated by a chill which may or may not have followed exposure. After a few days the râles disappear as consolidation becomes complete in the affected part, and the area which gave impaired resonance on percussion now gives a dull note, while the bronchial breathing in the affected part becomes more marked. The lips are apt to be attacked by herpes. With the fall of temperature, or crisis, which may be reached by the third to the ninth day, the râles return (*râles redux*) and become more and more loose, coarse, and moist as resolution progresses, until the lung becomes entirely clear, and only a slight roughening of the breath-sounds is to be heard. Bad symptoms in such a case are delirium, a feeble pulse, a feeble heart with distant heart-sounds, or one in which the action is labored and irregular. Prune-juice sputa, or, as the disease progresses, purulent sputa, are bad signs also. If the temperature falls to normal about the fifth day and then rises again, forming a pseudo-crisis, the attack will probably be prolonged. When a child is affected by croupous pneumonia it is very common for us to find all the ordinary objective symptoms without any of the physical signs just named. The dulness on percussion is difficult of development, because the chest is so resilient that the percussion blow makes the whole chest resound, and it is noteworthy that percussion of the chest on the diseased side quite commonly develops a high-pitched tympanitic note such as we often find above a pleural effusion.

Care should be taken in all suspected cases of croupous pneumonia that another common cause of the same symptoms does not mislead the physician. This cause or condition is acute pneumonic phthisis. In many cases only the finding of tubercle bacilli in the sputum, and the fact that resolution is delayed, and that finally the lung breaks down and forms a cavity, will permit a diagnosis of acute tuberculosis to be made, although the profuse sweats and rapid loss of weight may before this reveal the true state.

There are two areas in the lung often affected very early in pneumonia, particularly of the croupous type, and in pulmonary tuberculosis, which are apt to be overlooked, namely, the axilla and the septum between the upper and middle lobe on the right side, an area exposed to percussion and auscultation only when the right hand of the patient is placed on top of his head in such a way that the angle of the scapula is drawn away from the vertebral line. (Fig. 114.) If this is done, the inner border of the scapula will approximate the line of the septum, and along this line there will often be found in tuberculosis of this portion of the lung marked dulness on percussion or, on auscultation, râles, and the other physical signs of consolidation, even though the physician is unable to find elsewhere any evidence of local disease to account for the general systemic symptoms. Very often careful auscultation of the axillary area will also reveal signs not to be found elsewhere, which account for the illness, such as those of pneumonia or pleurisy, for here, as a rule, the friction-sounds of the latter affection are best heard.

FIG. 114.



Area of dulness found in many cases of obscure pulmonary tuberculosis, when the arm is raised so that the scapula no longer covers the septum.

There is another state that gives dulness on percussion, crepitant râles, and the other physical signs of pneumonia, namely, pulmonary congestion dependent upon the action of a feeble heart in the course of prolonged exhausting fevers or mitral disease; but the history of the illness, the feeble heart, and the development of these signs in the dependent parts of the chest effectually preclude the idea of any acute inflammatory process in the lung.

Finally, we frequently have after a pulmonary apoplexy an area of consolidation in the lungs; but if this be the case, we also have, as a rule, a history of recent hæmoptysis. This condition is, however, comparatively rare.

The condition of croupous pneumonia cannot readily be confused with any other disease except acute pneumonic phthisis, because of its characteristic symptoms, but catarrhal pneumonia and tuberculosis of the lung often are confused.

IN CATARRHAL PNEUMONIA the patient usually presents a history of recent illness of which the pulmonary state is a sequence. The disease rarely begins with the marked and startling symptoms of the croupous form, but is insidious and accompanied by a milder but more prolonged fever. Percussion often will not give the positively dull note which can be elicited in croupous pneumonia, and only impairment of resonance may be developed. There are increased vocal fremitus on palpation and increased vocal resonance on auscultation; there are also increased bronchial breathing and more bronchial râles than in the croupous form, for the disease is a broncho-pneumonia involving the bronchial tubes and adjacent tissues. The signs are generally diffuse, very often heard best at the bases posteriorly, and clear tubular breathing, such as is heard in the croupous form, is rarely to be found. The sputum is not sticky or rusty; the fever does not end by crisis, but rather by lysis; and the lung returns to its normal state very slowly, its condition often remaining almost stationary for weeks at a time.

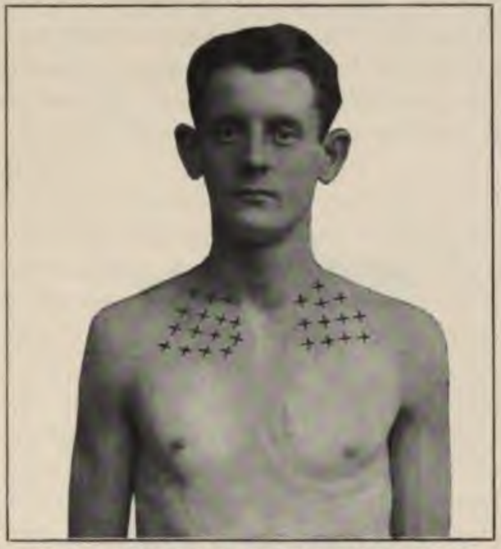
The separation of these symptoms of catarrhal pneumonia from those of early pulmonary tuberculosis is practically impossible by the physical signs until the case has progressed to a well-advanced stage, and the sputum should be well watched for tubercle bacilli.

Pulmonary Tuberculosis. Often catarrhal pneumonia merges into tuberculosis, and very often the diagnosis of catarrhal pneumonia proves to have been made in a case in which the disease is really primarily tuberculosis.

We have to rest the diagnosis of tuberculosis chiefly on the family history, the personal history, the fact that recovery does not take place, and that the patient loses weight more or less rapidly, and, more important than all, the presence as the case becomes well advanced of tubercle bacilli in the sputum, and later on yellow elastic fibres which indicate a breaking down of the lung-tissues.

In other instances a patient after complaining of a persistent "cold" with more or less cough fails to get better, and suffers from loss of appetite, loss of weight, and develops chills and fever, also becoming more or less anæmic. In such cases it is particularly necessary that the physician be on the *qui vive* for the physical signs of pulmonary tuberculosis, as this is the stage in which the disease is still curable in some instances. In such patients a slight prolongation of expiration, with harsh inspiration, and impairment of resonance on percussion, may be the only, yet very important, physical signs.

FIG. 115.

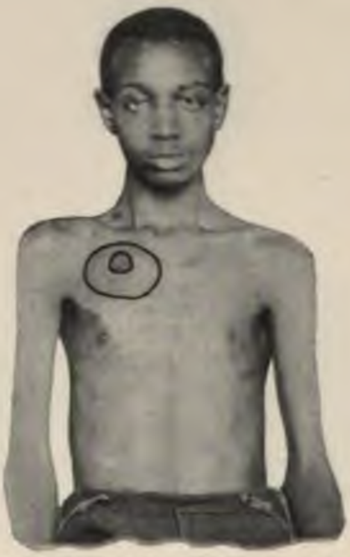


Areas of fine moist râles in early stages of tuberculosis of apices of lungs with impaired resonance on percussion and prolonged harsh expiration.

If the malady be tuberculosis and progressive, we soon find in the chest and sputum signs which make the diagnosis clear. The chest on inspection does not move with costal breathing as much as is normal; the hand placed upon it feels, when the patient speaks, that there is not only increased fremitus, but a bubbling feeling from coarse râles, and auscultation also reveals loose râles, the signs of the breaking down of lung-tissue. Finally, when a cavity is developed the percussion-sound over it becomes high-pitched, and, if the cavity be large, almost tympanitic, although all around it dulness may be present. If the cavity is large, it will often be found that it is possible to develop a still more tympanitic percussion-note if at the same time that the chest is percussed the patient holds his mouth open. This is called "Wintrich's change of note." The breath sounds now become more tubular or amphoric, and vocal resonance may be increased to such an extent that bronchophony or pectoriloquy becomes marked even in that part of the lung in which in health the vesicular sounds are heard most typically. (Fig. 116.) Prolongation of expiration is also present, and sweats, irregular hectic fever, and great loss of flesh ensue.

When there is fibroid phthisis of the lungs, inspection of the thorax often shows marked retraction of one side of the chest, more often the left side in front near the apex. The supraclavicular fossa and the costal interspaces are apt to be retracted, the scapula drawn nearer the spine, and that side of the thorax is poorly expanded on inspiration. The respiratory murmur is feeble and distant, and resonance on percussion is below normal. If secondary pleural contractions occur, the heart may be drawn from its normal position, and the other lung is apt to be emphysematous. This form of pulmonary tuberculosis is usually very slow and lasts for years.

FIG. 116.



Case of pulmonary cavity due to tuberculosis. The central ring is the area giving the physical signs of cavity, with cavernous breathing and whispering pectoriloquy, and the outer ring that of consolidation (dulness), with rapid breaking down of the lung-tissue (moist râles).

In striking contrast to the form just described is acute pneumonic phthisis, which often begins with the suddenness of an acute croupous pneumonia, with a chill, blood-tinged sputum, and pain in the chest. When it fails to undergo resolution the sputum is examined, and tubercle bacilli reveal its true nature. In other cases an attack of true croupous pneumonia sets free tubercle bacilli which have been walled off from the rest of the lung, and so precipitates acute pulmonary tuberculosis.

In some cases of suspected pulmonary tuberculosis the Röntgen rays can be used with great success in aiding difficult diagnosis. It is best to employ the fluoroscope rather than the radiograph, since with the former different parts of the chest can be rapidly compared, and the focus readily changed for deep and superficial tissues. Consolidated areas are dark and shadowy; cavities are light surrounded by shadow. So, too, abscess of the lung produces a distinct shadow in many cases.

Finally, it is to be recalled that mitral stenosis or even regurgitation, may, by producing engorgement of the lung, produce physical signs closely resembling pulmonary tuberculosis, for there are continued cough, dyspnœa, blood-spitting, and loose râles on auscultation. The absence of tubercle bacilli, and the presence of large cells filled with brown pigment in the sputum show the cause to be cardiac.

PULMONARY ABSCESS. The history of the case and its symptoms are our chief means of separating pulmonary abscess from pulmonary tuberculosis with the development of cavity, for the physical signs are about the same. In cases of abscess we find that the patient has suffered from pneumonia or from pyæmia with embolic infarction. In other cases discharges from the nose and throat entering the lungs produce such lesions. The symptoms of abscess, which separate it from cavity due to tuberculosis, are as follows: in abscess the lesion exists in the lower lobe, as a rule, while the tubercular cavity is usually found at the apex or in the upper lobe. The constitutional disturbance in abscess is often very slight, whereas in tuberculosis it is usually severe. In abscess the sputum is copious and purulent, and often coughed up in gushes, whereas in tuberculosis it is often scanty, and not markedly purulent, as a rule. Again, in the last-named disease tubercle bacilli may be found, but they are absent in abscess unless tubercular infection is simultaneously present.

The use of the fluoroscope is of great value in localizing the pus in some cases.

Pulmonary Gangrene. If the patient has the signs of cavity of the lung, and in addition an exceedingly fetid breath, with great wasting, the case is probably one of pulmonary gangrene. Gangrene is usually found at the base of one lung, as is abscess. The sputum is usually brownish.

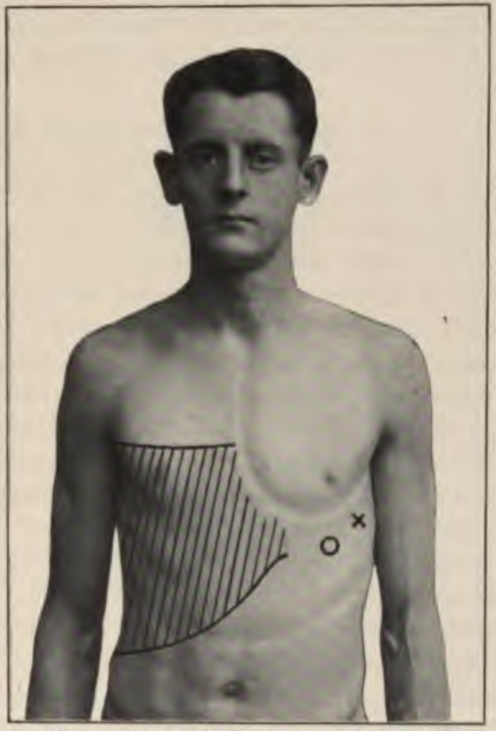
Bronchiectasis with fetid breath is occasionally met with, but the fœtor after coughing is never so exceedingly offensive as it is in cases of gangrene.

Pulmonary Œdema. The physical signs of pulmonary œdema may develop suddenly as a result of an injury to the vagus, or in acute disease of the lungs. Generally, however, their onset is slow and insidious, but the rapid breathing, the crepitant râles, the limitation of these signs chiefly to the lower part of the chest, the bilateral character of the signs combined with dulness on percussion, the absence of fever, the frothy sputum, and, it may be, the presence of renal or cardiac disease, all point to the true state of affairs. Such a state not rarely develops in the course of pulmonary congestion in old persons, particularly if influenza is the cause.

Pleuritis. To cite another form of thoracic disease, let us suppose that a healthy man is seized with pain in the thorax and a chill followed by fever. An examination of his thorax will reveal on inspection deficient breathing on the affected side, which is fixed because of pain produced by the inflamed pleural surfaces moving over one another on inspiration. Exaggerated breathing will be found on the opposite side to compensate for this fixation, and auscultation on the painful side will reveal a friction-sound, probably best heard in the axilla. (See Fig. 112.) After these signs have existed some hours the second stage develops, and as effusion take place we find that the friction-sound disappears, and that the affected side, previously almost normally resonant, is beginning to become dull, and, finally, is flat on percussion at the most dependent part of the pleural sac, namely at the base of the lungs posteriorly. This area of flatness on percussion gradually rises higher and higher until the effusion is completed. It extends anteriorly, and may be demonstrated as well here as it can be posteriorly and laterally, although, if the patient lies on his back or is partly recumbent, the entire anterior surface of the chest may be resonant, owing to the fluid leaving the front of the chest and going to the more dependent parts. In other words, in cases of non-sacculated serous pleural effusion changes in the position of the patient cause alterations in the area of flatness on percussion, unless the effusion is large enough to fill the chest entirely, when, of course, it is immovable. Inspection will show an increase in the size of the chest on the diseased side, with bulging of the intercostal spaces.

A curious yet important point in this connection is the fact that the line where flatness on percussion ceases at the top of the effusion posteriorly is wavy or sigmoid (S-shaped). Above the level of the effusion percussion over the compressed lung gives a somewhat hollow note or hyperresonance, called "skodaic resonance," and the sense of resistance to the percussed finger is less at this point than over the effusion where the resistance is great. Percussion and palpation will reveal the lower margin of the liver depressed if the effusion is on the right side (see Fig. 117). In auscultating the

Fig. 117.



Right-sided pleural effusion. The area of flatness on percussion in the thorax merges with that of liver dulness, and the lower border of the liver is below its normal level. The apex-beat is displaced to the left at *x*. The circle represents Traube's semilunar space, which is obliterated in left-sided effusion but not in right-sided effusion.

chest in the area in which flatness has been developed by percussion the breath-sounds are very indistinct, except in the back near the vertebral column, where there may be marked blowing breathing. If the patient speaks, there will be found loss of vocal resonance and of fremitus over the effusion, but along the margin of the spine on the diseased side there may be heard in some cases bronchophony, or even the bleating voice-sound called ægophony. Inspection and palpation will show the apex-beat of the heart displaced to the right and downward in cases of effusion into the left pleura, and to the left in cases of right-sided effusion. Again, if the effusion be on the left side, it will be found on percussing "Traube's semilunar space," a space directly in the mamillary line and a little below the nipple, that the usual tympanitic resonance normally found in this area is extinguished through the downward pressure of the fluid.

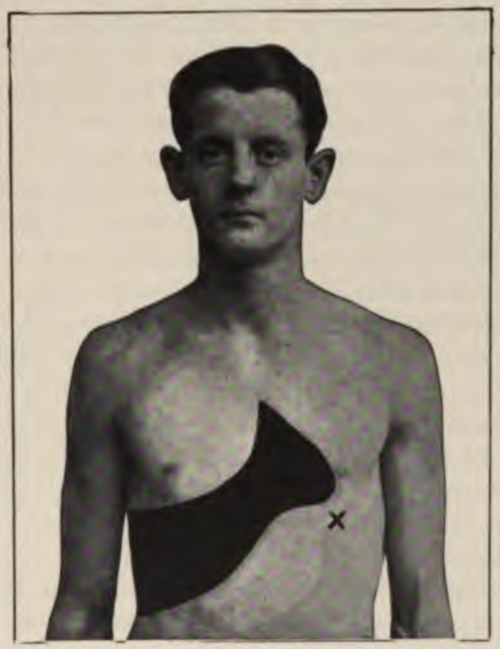
If the effusion be accompanied by pneumothorax (see next page), we will find three sets of physical signs, namely, those of effusion, which will be at the lowest part of the chest, next above this an area in which percussion gives a clear tympanitic note due to the air in the pleural cavity, and above this the physical signs of the compressed lung in the apex of the chest cavity. In this condition we may also hear succussion or splashing sounds, if the patient is shaken while the physician's ear is against the chest-wall, and the metallic tinkling, or dropping sounds, as the fluid falls from the top of the chest-cavity into the effusion. Again, we may use what has been called "coin percussion." This consists in having an assistant place a large silver coin against the chest-wall on the diseased side anteriorly, and then the physician listens at the posterior aspect of the chest, his unused ear being closed by his finger. The assistant now strikes the silver coin with the edge of another silver coin. If the coins be struck together below the surface of the effusion, very little of the metallic sound will be transmitted through the chest. If the coins are struck together at the level of the layer of air, the sounds come through the chest-cavity with startling clearness; but if at the level of the lung, they are less clearly heard than at the level of the air, but more so than at the level of the effusion.

The reasons for this are obvious, for the liquid prevents transmission of the metallic sounds, as does also to some extent the compressed lung at the apex of the chest, whereas the space filled with air conveys the sounds directly to the ear.

Finally, if the effusion is absorbed by unaided nature, the area of flatness on percussion becomes less and less from above downward, the expansion of the chest on inspiration increases, the interspaces cease to bulge, and the friction-sounds may return for a brief period.

If the effusion is purulent, the patient is apt to lose flesh and strength, to have chills, fevers, and sweats, and to present all the evidences of an accumulation of pus in some part of the body. Particularly is this result apt to follow a pleurisy complicating one of the acute infectious diseases, such as scarlet fever, typhoid fever, pneumonia, and in many cases in which tuberculosis is responsible for the illness. In children empyema is not so serious as it is in adults. In the former it is usually due to the pneumococcus, in the

FIG. 118.



Showing at *x* mark the so-called area called Traube's semilunar space, where in health percussion gives a tympanitic note, which becomes flat in left-sided pleural effusion. The solid block represents hepatic and cardiac dulness.

latter to the streptococcus. The following statistics, compiled by Netter, show the frequency with which these organisms produce empyema in adults and children:

	Children.	Adults.
Pneumococcus	53.6	17.3
Pneumococcus and streptococcus	3.6	2.5
Saprophytic organisms	10.7	. .
Staphylococci	. .	1.2
Tuberculosis	14.3	25.0
Streptococci	17.6	53.0

Empyema may also be due to the bacillus typhosus, the colon bacillus, the micro-organism of influenza, the gonococcus, and actinomycosis.

Particular attention should be called to the possibility of pleural effusions coming on insidiously. There is probably no other massive pathological change anywhere in the body so often unsuspected or overlooked, and it is noteworthy that, when pleural effusion is insidious in its onset and devoid of prodromes, it is often due to an undiscovered tuberculosis, whether the exudate be found to be serous or purulent. Again, the fact that tubercle bacilli cannot be found in the effusion when it is aspirated in no way proves that the effusion is not tubercular in origin, since they are rarely found in the fluid even when tubercular pleurisy is most active.

Serous pleural effusion single or double may result from thrombosis of the vena azygos. It is particularly apt to come on in patients suffering from typhoid fever or other exhausting diseases. Such a transudation can be separated from the effusion due to inflammation by the method of Pohl and Rosenbach. This consists in withdrawing some of the fluid by an aspirating-needle after the patient has received a dose of iodide of potassium. A few drops of fuming nitric acid are added to the fluid, and it is then agitated with chloroform, when, if the effusion be a transudation, the iodine will be seen of a red color sinking to the bottom of the test-tube with the chloroform. If it be an inflammatory exudate, the iodine will not be passed into the effusion.

If on aspirating the fluid in the chest it is found to be hemorrhagic in character, the cause may be one of the diseases which produce marked asthenia, notably carcinoma, nephritis, one of the acute infectious diseases in a malignant form, or tuberculosis. The cancer may or may not be in the chest. Rarely such an effusion occurs in otherwise healthy men without these causes. The possibility of the hemorrhagic effusion being due to a leaking aneurism, or to leakage from an ulcerated bloodvessel in tubercular disease of the lung, is to be remembered.

Sometimes interlobar pleurisy develops and effusion of fluid or of pus takes place in such a position that it lies between the two lobes without escaping into the general pleural cavity. Under these conditions the symptoms of an ordinary pleuritis may be present, and the physical signs will consist of an area of flatness on percussion, which is sharply outlined, and is bordered *above* and *below* by an area of high-pitched resonance on percussion, and tubular breathing due to compression of the lung.

PNEUMOTHORAX. The development of sudden dyspnœa and thoracic pain, with pallor and cyanosis, a subnormal temperature and a rapid pulse, during the course of a case of tuberculosis of the lung in particular, should lead us to suspect pneumothorax. Inspection shows distention of the affected side, bulging of the intercostal spaces, and a sensation of distention of the chest to the physician's hand. The act of respiration moves the involved side but slightly, while the opposite side is moved greatly. The apex of the heart is displaced and percussion gives a loud hollow note. Auscultation of the affected side reveals absence of breath-sounds, and the resonance on percussion with absence of respiratory murmur makes a very pathognomonic combination of symptoms. Metallic sounds are often elicited in this condition arising from unknown causes. It will be found that if the pleximeter is struck with the handle of a percussion hammer, while the physician auscults the chest elsewhere, a clear distinct metallic sound is transmitted to the ear. When a pneumothorax communicates freely with a bronchus we often have gurgling- or bubbling-sounds due to bronchial secretion, or if pus is in the thorax, we find moist râles in the bronchial tubes and purulent expectoration. It is remarkable how differently patients suffer when affected by pneumothorax. Some are in the most urgent dyspnœa, while others after a very short time seem to be able to take some exercise without grave embarrassment.

BRONCHITIS. If after exposure to cold there is a sense of soreness in the chest, with more or less oppression and a hard cough, which seems to tear the bronchial tubes, the cough being without associated expectoration and the febrile movement but moderate, we suspect the presence of an acute bronchitis; a diagnosis which will be confirmed if we find the following physical signs:

There is marked roughening of the breath-sounds all over the chest, particularly over the bronchial tubes at the back, between the scapulæ, without any increase in vocal resonance and fremitus or any impairment of resonance on percussion. As the disease progresses these sounds of harsh breathing give way to râles, which are at first fine and moist, then coarse and sonorous, as the second stage, or stage of secretion, develops; and, finally, they decrease little by little, as health is approached and the mucus is expelled by coughing. Care should always be taken to determine in examining a case of suspected bronchitis that the symptoms are not due to a broncho-pneumonia.

Should the case become chronic, the sounds of coarse and more or less sonorous râles will persist and become constant. Such cases usually become worse in winter, and the sputum is sometimes very profuse (bronchorrhœa). The physician should always be careful in these cases to see to it that renal disease or a feeble heart is not the cause of the bronchial disorder. The health suffers but little in simple chronic bronchitis; but if bronchiectasis develops it may be much impaired.

Too often the careless diagnosis of chronic bronchitis is made when the trouble is due to pulmonary tuberculosis or a feeble heart.

Under the name "putrid bronchitis" we have a state in which the sputum is foul and expelled in a liquid form, in which float little yellow plugs (Dittrich's plugs). This condition may end in pulmonary gangrene or cause metastatic abscess.

EMPHYSEMA. The presence of a barrel-shaped chest, with almost immovable walls and marked abdominal breathing, points to the presence of emphysema of the lungs, and this opinion is confirmed if on auscultation of the chest we find *marked prolongation of expiration,* diminished vocal resonance and fremitus, and increased resonance on percussion. The face is often quite cyanotic, the superficial veins of the neck turgescent, the abdominal respiratory movements abnormally great, and the superficial veins in the epigastrium enlarged. If bronchitis or bronchiectasis is associated with the emphysema, as is frequently the case, we find more or less marked râles all over the chest, particularly posteriorly. Sometimes a systolic murmur can be heard over the tricuspid area, due to regurgitation on the right side of the heart. Cardiac dulness is generally obliterated by the enlarged lung, and the apex-beat cannot be felt except in the neighborhood of the ensiform cartilage or in the epigastrium. Both the hepatic and splenic dulness are found to begin and extend lower than normal, owing to the expansion of the lung. We may also find accentuation of the second sound in the pulmonary artery. The tricuspid regurgitation not rarely develops as a result of a damming up of the blood in the right ventricle.

When a patient is seized with a violent attack of dyspnœa its cause may be asthma, a foreign body in the air-passages, or laryngeal spasm.

ASTHMA. If it is asthma, there will be labored breathing in which all the accessory muscles of respiration in the neck and trunk aid the ordinary respiratory muscles. The posture of the patient will usually be that of sitting up in bed and somewhat leaning forward. The face will be flushed, the vessels of the face and neck turgid, and the lips may be cyanotic. Often the patient, while sitting up, supports himself by resting on his hands, which are placed at his side in order to raise his shoulders and fix the chest-walls for contraction of the muscles which are endeavoring to drive out the air, for it is to be remembered that the respiratory difficulty in asthma depends more upon the fact that the patient cannot empty the lungs than upon the fact that he cannot fill them. As a matter of fact, they are too full of air which has been used.

Inspection not only shows these signs in asthma, but also reveals, in cases in which emphysema has not developed to such an extent as to cover the heart with the lung, that the apex-beat is diffused and the heart laboring. Palpation reveals little except when coarse râles are present in large numbers, when some bubbling may be felt.

Percussion usually gives an increased resonance, because the chest is inordinately full of air, and auscultation reveals very loud blowing breathing, musical notes, or squeaking or creaking noises, both on inspiration and expiration. Finally, as secretion begins to be established, musical and cooing râles may be heard, in well-marked cases, all over the chest before the ear is placed against the patient. At first these râles are heard chiefly on expiration, but very shortly they occur equally loudly on both inspiration and expiration.

If on auscultating the chest we find it filled with musical and cooing râles heard in every part, although most marked in the bronchial tubes, we may be fairly sure that an attack of asthma is about passing away; but if, on the other hand, the attack is beginning, the manifest difficulty in breathing, the prolonged expiration, with comparatively few râles, the harsh bronchial sounds, and the general objective symptoms of the case will explain the cause of the pulmonary condition. Toward the end of the attack coughing brings up a limited amount of sputum, which contains Curschman's spirals and Charcot-Leyden crystals. (See chapter on Cough and Expectoration.)

As asthma is a symptom, not a disease in itself, the physician should always examine the nose, with the object of discovering some source of reflex irritation in the nasal mucous membrane, or test the urine to discover whether renal disease is present, or the heart to determine if a cardiac lesion accounts for the symptoms. Sometimes gastric disorder is responsible for the attack.

Care should be taken that a catarrhal pneumonia developing after an attack of asthma is not overlooked until the patient is dangerously ill.

FOREIGN BODIES IN THE AIR-PASSAGES. The dyspnœa due to a foreign body in the air-passages, whether it be a piece of meat or a false membrane, is quite different from that of true asthma, for in this case the difficulty is commonly in the entrance of air. The onset of the attack is usually sudden, but inspection will show that on inspiration the costal interspaces are greatly drawn in, as is also the epigastrium. There will be practically no signs in the chest which are not evidently due to the efforts at forced breathing, and a history of having had a foreign body in the mouth or of some laryngeal disease will usually be obtainable. Obstruction may, however, be present, and this history be absent in cases in which an abscess has burst into the air-passages from the mediastinum or through the posterior pharyngeal wall. In such a case, however, there would be, in all probability, purulent expectoration.

LARYNGEAL SPASM producing difficult breathing causes symptoms precisely like those of a foreign body in the larynx, except that in spasm the cough is often constant and is very brassy or ringing. The patient will show by a gesture with his hand that the obstruction is in the larynx, if unable to speak. Such obstruction when seen in children is, as a rule, due to spasmodic croup, and, if so, probably depends upon one of three causes, namely, laryngeal catarrh, rickets, or digestive disturbance. If in an older person, it is probably due to aneurism pressing on the recurrent laryngeal nerve, to a laryngeal crisis in locomotor ataxia, or to growths in the mediastinum producing pressure on the nerve-trunks going to the laryngeal muscles. Sometimes great enlargement of the retro-bronchial glands will cause laryngeal spasm or obstruction by pressure.

TUMORS occur in the chest generally as mediastinal growths, and are most commonly sarcomata or lymphadenomata. There will be found, if the growth be large, evidences of its pressure upon the chest-wall, such as bulging and dulness on percussion over the swelling. This level of dulness is unaltered by changing the posture, as it may be in pleural effusion. Generally there will be evidence of pressure on the bronchial tubes, which causes dyspnœa, and of pressure on the thoracic vessels, which produces signs of impaired circulation as shown by cyanosis, venous engorgement, and flushing of the skin of the face and neck. Often such growths cause pleural effusions by pressure on the bloodvessels, or produce pulmonary consolidation by causing an exudation in the lung-tissue.

The diseased conditions from which it is necessary we should distinguish mediastinal growths during life are as follows: 1. From aneurism. 2. From abscess. 3. From pleural effusion. And 4. From chronic pneumonia. There are several subdivisions of these diseases that might be made, but to all intents and purposes these are sufficient. Pericarditis may, perhaps, be named as the fifth lesion to be thought of. On general principles it may be said that primary mediastinal growths are so rare that they can be excluded on this ground in many cases.

Aneurism in the thorax is sometimes so extremely difficult of absolute diagnosis that but few rules can be laid down for its differential diagnosis from growths in the mediastinum, for deeply seated aneurism in this region cannot be said to possess any pathognomonic symptoms. The various portions of the aorta in which aneurism occurs make its symptoms different in almost every case, and we are forced to rely more upon general conditions than absolute signs. Thus, if a patient has no direct symptoms of aneurism, and none of those conditions present which we know predispose to such a lesion, such as atheroma of the bloodvessels, due to Bright's disease or any other similar cause, or syphilis, rheumatism, or a history of violent exertion or severe toil, we may with a certain degree of assurance look further for symptoms of mediastinal trouble of another sort than aneurism. (See Aneurism in this chapter.)

Unfortunately, the most common age for aneurism is much the same as that for mediastinal disease, although mediastinal disease seems to occur more frequently in youths than does aneurism, or, in other words, is scattered over a wider range of years. The pain of aneurism is generally considered to be more violent than that of any other thoracic lesion except angina pectoris. If the aneurismal sac be large enough to give us a wide area of dulness on percussion, there ought to be an expansile movement. Hæmoptysis is not in any way a differential sign, since in the one case it may be due to aneurismal leakage, and in another to ulceration of small blood-vessels by pressure exercised by a tumor, be it aneurismal or malignant, or even benign. Aneurism is relatively very frequent.

MEDIASTINAL ABSCESS is to be separated from mediastinal tumors with care. In the first place, in abscess we generally have a history of infection, or, if the case be one of cold abscess, it is commonly associated with a history of struma or spinal disease. If the abscess be acute, there is generally the history of pain, followed by a chill more or less severe, and fever; or, if cold, then we frequently have irregular febrile movements, with long-continued anorexia and loss of flesh. Cold abscess, too, is generally in the posterior mediastinum, while acute abscess generally occurs in the anterior space.

Pulsation may frequently occur, owing to the transmission of the aortic or cardiac impulses, and affords no better diagnostic point here than elsewhere. In some cases where the presence of aneurism is extremely doubtful and the likelihood of abscess extremely probable, an exploratory needle may be used, either through a hole drilled in the sternum or passed between the ribs; but a careful review of the history of the case should certainly always be made and used as a basis from which to draw conclusions.

By far the greatest difficulty may be experienced when we attempt to diagnosticate between pleural effusion produced by pleurisy and pleural effusion produced by mediastinal disease, provided the case be not seen from the first and the history be obscure. If the effusion be not great, we may be able to discern friction-sounds produced by the rubbing of the tumor against the chest-walls; but if the effusion be large, this sign may not be recognizable. All other methods failing, it would be advisable to tap the chest, and, if the fluid drawn be fibrinous, we know it to be inflammatory; while if it be clear and limpid, or at least thin and not viscid, it is probably due to pressure. This is not, however, a positive sign, since very frequently in cases of asthenic inflammation we have an exudate lacking entirely in the fibrinous constituents (see Pohl and Rosenbach's Test, page 290).

Tumors of the mediastinum invading the lungs have frequently been mistaken for chronic and even acute pneumonia, passing, as they do, along the larger bronchial tubes and bloodvessels.

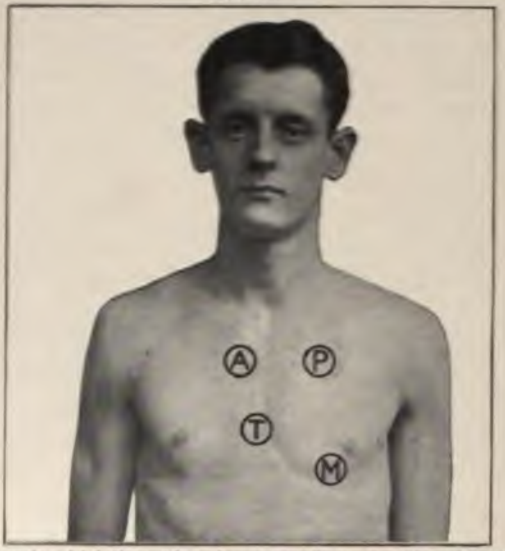
Without doubt, in a certain number of cases, either hypostatic pneumonia or pneumonia due to pressure on the bronchial vessels develops as the tumor invades the lung, and in such cases it is absolutely impossible to make a diagnosis unless by symptoms of pressure in the mediastinum, or some history pointing to such a result. Walsh has stated that if the lesion be due to a tumor, the affected side will increase in bulk rather than diminish, and that dyspnœa out of proportion to the degree of consolidation points to a mediastinal disorder rather than one confined to the lungs. If the heart be displaced in either direction, the odds point to mediastinal tumor; but the presence or absence of a hæmoptysis, as has just been stated, influences the diagnosis not at all.

The diagnosis of pericarditis from mediastinal lesions is much more readily made. The history of sudden præcordial pain and the limited area of dulness on percussion aid us very materially in deciding as to what the disorder is, while the description of the onset of the attack, with a few pointed questions as to systemic taints, etc., may do much to unravel the mystery. The distention of the pericardial sac from effusion may give us a regular outline on percussion, while the dulness of mediastinal disease may be irregular and varying.

Auscultation of the Heart and Vessels. On attempting to study the heart-sounds, we usually auscult the neighborhood of the apex-beat and expect to find, if the heart be healthy, two sounds, occurring one immediately after the other, which resemble the sound of the words "lub dup," the "lub" being the so-called first sound of the heart, produced by the contraction of the heart-muscle and the tense valves, and the "dup" being chiefly caused by the slapping to of the aortic valves. After listening in this region we next place the ear over the second right costal cartilage, in order to come as near as possible to the point of greatest intensity of the sound, produced by the aortic valves. If the heart is normal, we find only these sounds, "lub dup," and nothing else. If it is feeble from exhausting disease, from fainting, or by reason of fatty degeneration, we find that the sound "lub" is feeble, and the "dup" sound is also feeble, because the valves do not slap back into place with as much force as is normal. If, on the other hand, the heart is hypertrophied or stimulated, we find these sounds accentuated, and it is of importance to remember that marked accentuation of the aortic second sound, showing forcible closure of the aortic valves, indicates a condition of high arterial pressure, often the result of vascular spasm arising from chronic contracted kidney or general arterial sclerosis. On the other hand, if the pulmonary second sound at the third left intercostal space is accentuated, it indicates an increase in pulmonary pressure due to impediment to the flow of blood in the lungs. This pulmonary second sound is markedly accentuated in both mitral obstruction and regurgitation and in some cases of pneumonia and emphysema.[1]

The sound produced at the various orifices of the heart are heard best at or near the following points (Fig. 119), although the actual positions of the valves are shown in Fig. 120. The mitral valve is heard best at the apex-beat; the aortic valve at the second right costal cartilage, the tricuspid valve over the sternum on a line drawn

FIG. 119.



Showing the areas in which the various heart-sounds are best heard in health. A is the area for the aortic valve; P, that for the pulmonary valve; T, for the tricuspid valve; and M, for the mitral valve. The pulmonary circle is a little high.

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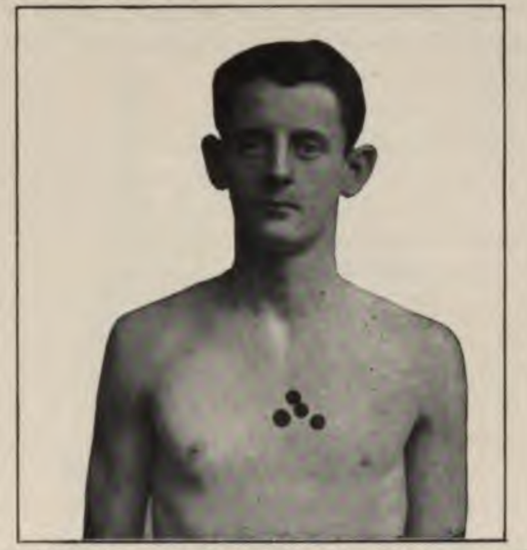
[1] By accentuation, I do not mean necessarily any increase in the loudness or volume of the sound, but an increase in its sharpness.

from the third left intercostal space to the fifth right costal cartilage, and the pulmonary valve at the third left intercostal space. All the heart-sounds may be reduplicated in health and in disease. When the first sound is reduplicated, it probably is due to asynchronism of the ventricles, and when the second sound is doubled there is asynchronous tension of the two sets of sigmoid valves. If disease of the valves be present, we are apt to find reduplication of the second sound in cases of mitral stenosis and in pulmonary disease producing an abnormally high tension in the pulmonary circulation. Such reduplication is also seen in some individuals suffering from aortic stenosis.

FIG. 120.



Approximate positions of the valves of the heart. To the left the mitral, in the centre the pulmonary, to the right the tricuspid, uppermost the aortic.

Compare with this the figure showing where the sounds of these valves are actually best heard, Fig. 119.

The Significance of Heart Murmurs. Supposing that on listening to the heart in the mitral area—that is, in the neighborhood of the apex-beat—there is heard in place of the normal sounds ("lub dup"), or with them, a murmur. What does it mean? It means that, friction-sounds being excluded, either valvular disease, aneurism of the aorta, or marked anæmia is present. Still more rarely the sound may be due to what is called a cardio-pulmonary murmur, which may occur at any time in the cardiac cycle. This sound is produced not by the movement of the lung in the respiratory act, but by the movement of the lung by the action of the heart, a movement which occurs approximately seventy times a minute, instead of fourteen times, as it would if respiratory. The sound is produced, not in the heart, but by the displacement of air in the lung, and the murmur can often be arrested or altered in character by changing the posture of the patient. The absence of any signs of cardiac difficulty, such as are met in true cardiac disease, and the fact that holding the breath on expiration or inspiration stops the murmur, aid us in suspecting that the murmur is one of the cardio-pulmonary type, particularly as it is apt to be musical in character.

The anæmic murmur is particularly apt to be heard in the case of a feeble child suffering from chorea, or, if in an adult, in association with other signs of disorder of the blood, which should make the physician suspect this condition to be the cause. Further than this, an anæmic murmur is apt to be soft and purring, and associated with rather feeble heart-sounds, probably due to the fact that the heart muscle is not well nourished; such a murmur will generally be found most marked at the left margin of the sternum near the third interspace.[1] (See chapter on Blood for illustration.)

Having found that there is a murmur, and having excluded the causes just named, it is now necessary to determine at what orifice of the heart it is produced, and the rule is to be remembered that a murmur is always heard loudest at about its point of origin. We therefore place the ear over the aortic cartilage (second right). If the murmur be mitral in origin, it will not be heard at this place, unless it be so loud as to be transmitted from the apex. If it is aortic in origin, it will be louder here than at the apex. If it is tricuspid, it will be loudest in the tricuspid area; if pulmonary, loudest at the pulmonary area. As murmurs at the tricuspid and pulmonary valves are rare, we nearly always have to deal with aortic or mitral murmurs, or both. In this way, therefore, we can determine the origin of the murmur, and that it is a mitral or an aortic murmur.

Let us suppose that it is mitral. We must determine whether it is that of mitral regurgitation or obstruction, or, as they are also called, incompetence and stenosis. The probabilities are that it is the regurgitant murmur, because this is by far the most common murmur heard in the heart; and if to this probability we add the fact that it is transmitted well into the axilla, and even heard at the angle of the scapula, our diagnosis is greatly aided, for this is the area of transmission of the murmur of mitral regurgitation. The most important diagnostic point, however, is the discovery that the murmur occurs simultaneously with the first sound of the heart, or with systole—that is, with the apex-beat or the carotid pulse. If it does, and the other signs of mitral disease are present, it is almost certainly one of mitral regurgitation. This murmur occurs with the first sound, or systole, because the ventricle in contracting drives most of the blood in the normal direction into the aorta, and also forces some of it back through the left auriculo-ventricular orifice into the auricle, causing a regurgitant murmur (Fig. 121). Sometimes there will be found in such cases a very marked accentuation of the second sound at the pulmonary orifice, due to the increased

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[1] It must not be forgotten that murmurs due to endocarditis also are frequently found in choreic children.

FIG. 121.

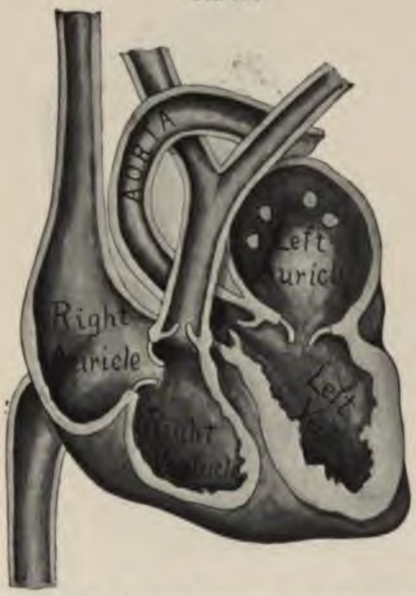
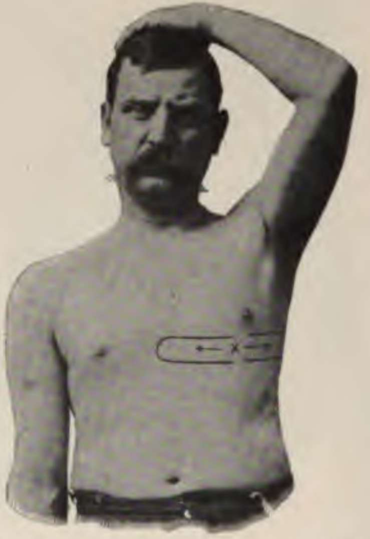


Diagram modified from Page to show the relation of the various valves. A study of this diagram will render clear the time of the various cardiac murmurs. Thus in mitral regurgitation the blood passes back from the left ventricle to the left auricle during systole, and is dammed up in the pulmonary veins, the openings of which are seen in the auricular wall, producing pressure on the pulmonary valves, the sounds of which are thereby accentuated.

pressure in the pulmonary veins by reason of the blood, which has been dammed back into them by the distention of the auricle due to the blood which regurgitates into it. The area of greatest intensity of the mitral regurgitant murmur is shown in Fig. 122.

In adults inspection and palpation will rarely reveal much of a thrill over the præcordium in mitral regurgitation, but in children this thrill is rarely absent and is usually well marked. Percussion will show that the area of cardiac dulness (see earlier part of this chapter) is broadened, extending beyond the right edge of the sternum and to the left of the mammillary line.

FIG. 122.



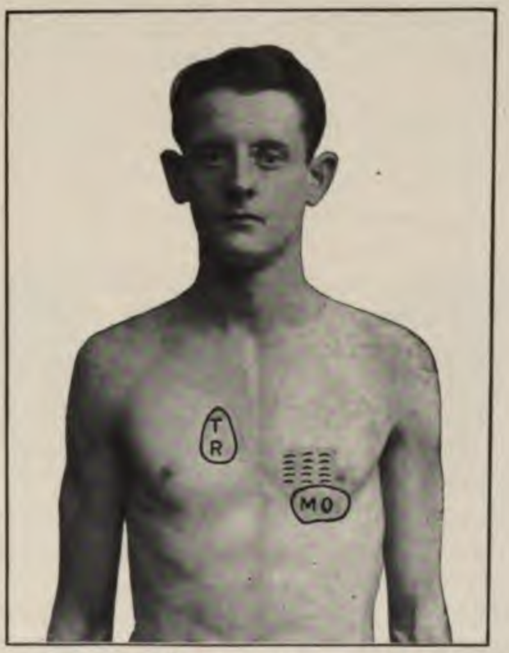
Showing at x the apex-beat where the murmurs of mitral regurgitation and obstruction can be best heard. The arrow pointing to the axilla indicates the direction in which the regurgitant murmur is transmitted, and the arrow pointing to the sternum the direction of transmission of the obstructive murmur.

If the pulse is irregular and asynchronous with the heart-beats, the heart enlarged by dilatation, and the urine scanty, we recognize that compensation is lacking and treatment needed.

In the diagnosis of the mitral regurgitant murmur the physician must not be misled by a loud aortic systolic murmur transmitted down the sternum to the area of the apex-beat.

If, on the other hand, the murmur is due to mitral stenosis, it will be found that it does not occur with systole, but just before it, and is not transmitted into the axilla, but to the right, over to the midsternal line, and it is presystolic in point of time. (See Figs. 122 and 123.) This murmur can often be exaggerated by placing the patient in a prone position, and occurs before systole, or the first sound, because it is produced by the blood passing through an obstructed left auriculo-ventricular orifice, and, as the ventricle does not contract (systole) until it is filled, the murmur must be made while it is filling, and so is presystolic in time. Palpation of the præcordium in such a case will usually reveal a marked diastolic thrill in the fourth or fifth interspace. If the compensation of the heart in a case of mitral stenosis is broken, these signs are accompanied by a very irregular action of the heart, the first sound becoming accentuated and the murmur disappearing or being inconstant, because the auricle is too feeble to drive the blood forcibly through the orifice. In some cases what is called a "gallop rhythm" develops, the heart-sounds being

FIG. 123.



MO shows area of greatest intensity of a mitral obstructive murmur. TR shows area of greatest intensity of a tricuspid regurgitant murmur; both lesions were present in this patient. The fine lines indicate the area in which is felt the characteristic thrill of mitral stenosis. (From the author's wards.)

reduplicated in such a manner as to make a sound somewhat resembling that made by a horse's feet when it is galloping. If in addition to these physical signs the physician finds cyanosis, jugular distention, congestion of the lungs, and dyspnœa, indicating a failing heart, for the venous system is engorged and the arterial system starved, the diagnosis of mitral stenosis with failure of compensation is established.

In the diagnosis of mitral obstruction the physician must not be misled by the possible presence of what is known as Flint's murmur, a presystolic murmur heard in the mitral area, and due to relaxation of the mitral valves, which are thrown into vibration during diastole by blood regurgitating from the aorta in aortic regurgitation.

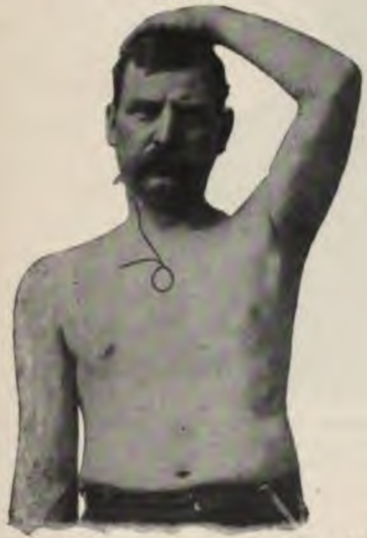
If, however, we have found the murmur to be aortic in origin, we must determine whether it is that of aortic regurgitation or obstruction or aneurism. The former condition is so common that it must be excluded from the diagnostic possibilities before any further steps are taken.

This murmur occurs with the systole of the ventricles, and the carotid pulse and apex-beat; it is harsh, as a rule, and is transmitted up into the carotid, and it may be into other arteries of less importance. (See Fig. 124.) It is produced by the contraction of the ventricle driving the blood through a narrowed or roughened aortic orifice. Considerable hypertrophy of the left ventricle is usually produced, and the apex-beat is strong and forcible if compensatory hypertrophy is present. A similar murmur may arise from vegetations on the aortic valves, or, in other instances, atheromatous changes in the aorta may produce a systolic aortic murmur.

As a matter of clinical fact true aortic obstruction, due to vegetations on or contractions of the aortic valves themselves, is always associated with a certain degree of aortic regurgitation, even though the regurgitant murmur may not be discoverable. Aortic systolic murmurs entirely free from an aortic regurgitation are really due to aortic roughening produced by atheromatous plaques on the aortic wall.

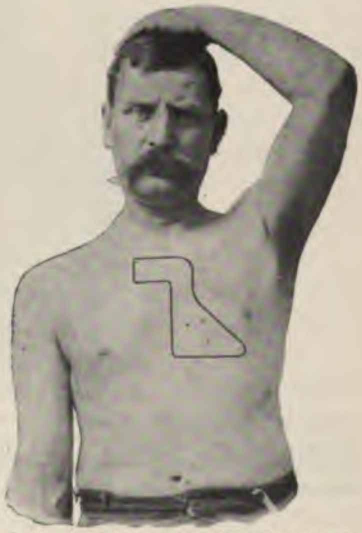
If, on the other hand, the murmur occurs after the systole or the apex-beat and is aortic, the murmur is that of aortic regurgitation, and is called the diastolic aortic murmur. It is heard loudly at the aortic cartilage, often equally marked to the left of the sternum, and it is transmitted down along the sternum very clearly and into the left ventricle, so that it is plainly heard at the apex. (See Fig. 125.) In this condition we have usually marked dilatation of the heart with hypertrophy (the so-called "ox-heart"), and a peculiar trip-hammer pulse (see chapter on Pulse), sometimes called the "water-hammer" or Corrigan pulse. This murmur is due to incompetence of the aortic valves, which allow the blood to regurgitate into the heart after it is driven out into the aorta. If in association with this murmur we find irregularity of the action of the heart, a lack of sharpness in its sounds, displacement of the apex-beat downward and to the left, extension of cardiac dulness to the right, and a feeble pulse, then we know that the heart is failing. Finally, a soft mitral systolic murmur and the development of râles in the chest at the base of the lungs show still greater failure, the result of dilatation of the mitral orifice, which is the result of the strain produced at this area by the constant regurgitation and consequent dilatation of the left ventricle. The objective symptoms of valvular disease are described later on.

FIG. 124.



Showing the area of greatest intensity and the direction of transmission into subclavian and carotid arteries of the aortic obstructive murmur.

FIG. 125.

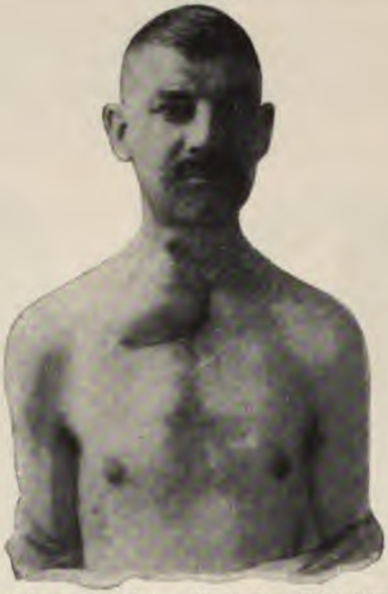


Showing the area in which the murmur of aortic regurgitation can be most clearly heard.

The characteristic symptoms of aortic aneurism vary greatly with the site of the lesion, as will be pointed out below. The most typical signs are a "bruit" or angry murmur, systolic in point of time, thrill over the growth, dulness on percussion over the area of this thrill, dyspnœa, cardiac hypertrophy, and functional disturbance of the heart. Finally, a history of alcoholism, syphilis, and severe strain or injury will be a valuable indication of a causative factor. It is to be remembered that small aneurisms deeply situated, which do not press upon other organs, may produce no symptoms for years, and finally be discovered only at autopsy. When the ordinary signs of aneurism are not clear, an examination of the radial pulses may reveal that one of them is delayed, and this is a confirmatory sign of aneurism.

Although the general symptoms of aortic aneurism have just been described, there are others which depend upon the seat of the aneurism, and which materially modify the importance of the points so far named in diagnosis. Let us suppose that a patient presents himself with great engorgement of the vessels of the head and neck and arm of the right side, with perhaps œdema of that arm. The heart may be pushed downward and to the left and the voice may be lost or partially impaired by pressure on the recurrent laryngeal nerve of the

FIG. 126.



Case of aortic and innominate aneurism, with erosion of the clavicle and ribs, from the author's wards in the Jefferson Medical College Hospital. This case is of extraordinary interest because this picture was taken thirty-five months after an arrest of the growth of the aneurism by electrolysis.

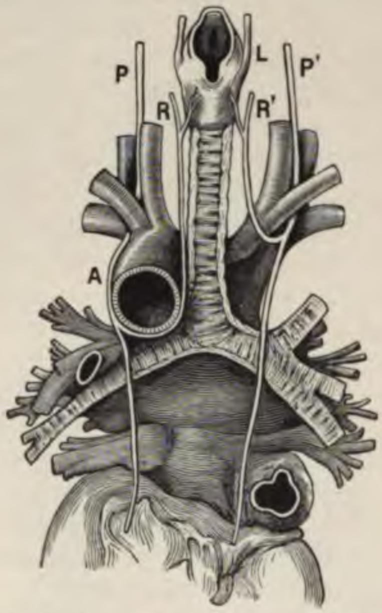
right side. The pupil of the eye may be widely dilated through irritation of the sympathetic, and there may be unilateral pallor of the face from this cause. If the pupil is contracted, then the ciliospinal fibres are paralyzed by pressure. In such a case pain is apt to be a prominent symptom and so severe as to be almost like that of true angina. Percussion over the second right interspace will give impaired resonance, and auscultation of the area of the pulmonary valve may show a pulmonic systolic murmur, due to pressure on the pulmonary artery, which in turn causes hypertrophy and dilatation of the right ventricle. There may be bulging of the first, second, or third interspace on the right side. Generally such symptoms will be due to an aneurism of the greater curvature of the ascending aorta, although they may be due to a tumor in the anterior or middle mediastinum; but the expansile pulsation, the bruit, and the history of the case will usually make the differentiation possible.

Again, let us suppose that the patient has a ringing, brassy cough, difficulty in swallowing, and expansile pulsation in the episternal notch and epiclavicular space of the left side, and dulness on percussion over the first and second left intercostal spaces. The left side of the face and neck may be engorged from pressure on the left innominate vein. Pupillary symptoms similar to those already named may be present. There is difficulty in breathing, particularly on inspiration, owing to the pressure of the growth on the trachea, the paralysis of the left vocal cord, and the pressure on the left bronchus, and there is dysphagia due to pressure on the œsophagus. The voice is altered from paralysis of the vocal cord due to pressure on the left recurrent laryngeal nerve. (See Fig. 127.) These symptoms indicate a lesion of the transverse arch.

If, however, none of these important signs are present anteriorly, we must search for some of them posteriorly, particularly the bruit and the expansile pulsation, and, if these are found to the left of the vertebral column opposite the angle of the scapula, we can rest assured that the aneurism involves the descending aorta. Severe intercostal pain is often felt in these cases. In this connection it is well to recall the fact that aneurism of the ascending portion of the aortic arch is by far the most common condition. Out of 953 cases of aortic aneurism, analyzed by myself and one of my assistants, Dr. Holder, no less than 570 were situated in the ascending part of the arch; of these, 544 were sacculated and 26 fusiform. Aneurism of the transverse arch occurred 104 times, and of the descending part 110 times. Of the 544 cases, 400 were in males.

There are other symptoms connected with aneurism which should not be overlooked. The first of these is "tracheal tugging," a sign which is found in some cases but not in all. The patient being in the erect position, the fingers of the physician grasp the cricoid cartilage and gentle upward traction is produced. If aneurism is present, a distinct tug will be felt with each beat of the heart.

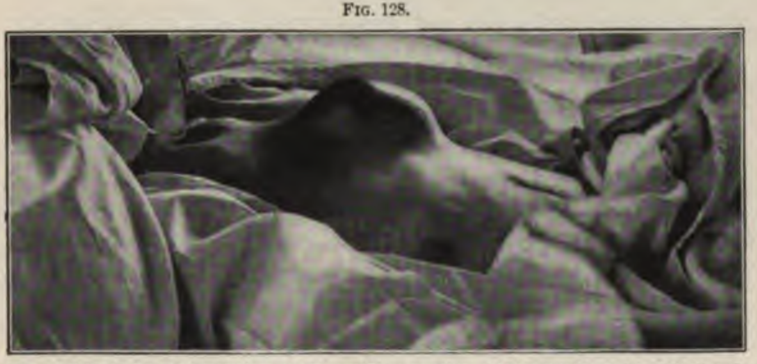
FIG. 127.



A posterior view, showing how an aneurism of the transverse aorta presses on the recurrent laryngeal nerve. The cut shows the relation of the aorta, where it is surrounded by the loop of the recurrent nerve, to the left bronchus and the lower part of the trachea. A, aorta; p, pneumogastric nerve; r, recurrent nerve; L, larynx. (DIEULAFOY, *Clinique Médicale*, 1896.)

It is important for us to remember, however, that this sign of aneurism is not pathognomonic, for, as Sewell has pointed out, it may occur in patients who have adhesions of the left pleura or diminished extensibility of the lung, or both combined. Further, in healthy persons, the descent of the heart with the diaphragm on deep inspiration may press the aortic arch on the left bronchus, and so transmit to the trachea a tug not due to aneurism. Another sign emphasized by Osler is the loss of pulsation in the peripheral vessels, the result of the loss of the heart's impulse in the aneurismal sac.

FIG. 128.



Anterolateral view of aneurism of descending portion of the aortic arch. At the dark spot at the apex the skin was ecchymotic from the near approach of the blood to the surface. (Private case.)

Doubtful cases of aortic aneurism should be subjected to the use of the fluoroscope, since this will often reveal the growth, particularly if the sac contains a firm clot.

If the examination has shown that the murmur is loudest in the tricuspid area, it is to be remembered that in the vast majority of cases the condition is one of tricuspid regurgitation, for tricuspid stenosis is an exceedingly rare lesion. The time of the murmur of the tricuspid regurgitation is identical with that of the mitral regurgitation (systolic), because the tricuspid valves are the counterpart in the right side of the heart of the mitral valves in the left.

Actual disease of the pulmonary valve is exceedingly rare, and usually congenital; pulmonary regurgitation is almost never met with. The signs of pulmonary stenosis due to congenital defect are cyanosis, hypertrophy of the right ventricle, a systolic murmur at the left side of the sternum, which is not transmitted upward, and a weak pulmonary second sound. The murmurs sometimes heard, and the thrills sometimes felt, in the pulmonary area are generally due to anæmia, the puerperal state, or some neurosis, or to congenital narrowing of the pulmonary artery, or to compression of the vessel by the heart. If the last two causes are present, the ventricular septum is usually deficient and cyanosis is noticeable.

In the diagnosis of all murmurs in the heart we must remember that several orifices may be diseased, producing associated murmurs. Some discussion as to the relative frequency of these associations has arisen, but the results of H. J. Smith derived from the London hospitals are usually accepted as correct. His results are as follows, in the order of frequency and association:

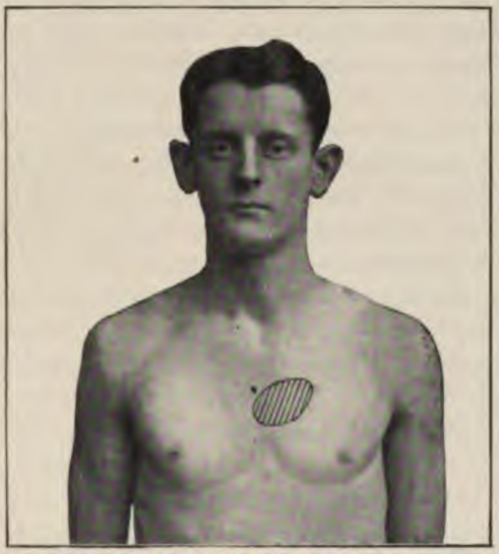
1. Aortic regurgitation and stenosis and mitral regurgitation.
2. Mitral stenosis and regurgitation.
3. Aortic stenosis and mitral regurgitation.
4. Aortic regurgitation and mitral stenosis.
5. Aortic regurgitation and stenosis.
6. Aortic regurgitation and stenosis; mitral stenosis and regurgitation.
7. Mitral regurgitation and tricuspid regurgitation.
8. Aortic regurgitation and stenosis; mitral regurgitation; tricuspid regurgitation.
9. Mitral stenosis and regurgitation; tricuspid regurgitation.
10. Aortic stenosis; mitral stenosis and regurgitation.
11. Aortic regurgitation; mitral stenosis and regurgitation.
12. Aortic stenosis; mitral regurgitation; tricuspid regurgitation.
13. Aortic regurgitation and stenosis; mitral regurgitation; pulmonary regurgitation.
14. Aortic stenosis and regurgitation; mitral stenosis.
15. Aortic regurgitation; mitral stenosis.
16. Aortic regurgitation; mitral regurgitation; tricuspid regurgitation.
17. Mitral stenosis; tricuspid regurgitation.
18. Aortic stenosis; mitral stenosis and regurgitation; tricuspid regurgitation.
19. Aortic stenosis; mitral stenosis.
20. Aortic regurgitation and stenosis; mitral stenosis and tricuspid regurgitation.
21. Aortic regurgitation; mitral stenosis and regurgitation; tricuspid regurgitation.
22. Aortic regurgitation and stenosis; mitral stenosis and regurgitation; tricuspid regurgitation.
23. Aortic regurgitation and stenosis; mitral stenosis and regurgitation; tricuspid stenosis and regurgitation.
24. Aortic stenosis; pulmonary stenosis.
25. Aortic stenosis; mitral stenosis and regurgitation; tricuspid stenosis and regurgitation.
26. Mitral stenosis and tricuspid stenosis.

The relative gravity of heart-lesions is, according to Walsh, as follows, the least dangerous being placed last and the most dangerous first:

Tricuspid regurgitation.
Mitral obstruction and regurgitation.
Aortic regurgitation.
Pulmonary obstruction.
Aortic obstruction.

Pericardial friction-sound is, of course, heard best in the præcordium at the base of the heart—that is, at about the third rib. It is separated from pleural friction by its frequency and by the fact that it continues when the patient holds his breath. (Fig. 129.)

FIG. 129.



Area in which pericardial friction-sound is best heard.

Laennec likened this friction-sound to the noise made by the leather of a new saddle when used for the first time. Sometimes it sounds like the crunching of dry snow under the shoe. It is usually a to-and-fro sound.

The general symptoms, subjective or objective, which a patient suffering from the various forms of valvular lesion presents, in some instances, have not been spoken of up to this point, because it is to be distinctly understood that murmurs produced by any form of valvular lesion may exist with great intensity without there being any systemic disturbance or the patient being conscious of their presence. On the other hand, the murmur may be so faint as to be almost indistinguishable, and yet the general symptoms of heart disease be very marked. This is because the development of general symptoms depends entirely upon the question of compensation by hypertrophy. If there is a leak in a valve or a constriction of an orifice, this leak or obstruction must be overcome by compensatory hypertrophy of the heart-muscle. If the heart-muscle can make up for the regurgitation or obstruction by increased effort, the circulation is unimpaired; but if it cannot do so, we have developed more or less rapidly, according to the lesion present and the condition of the heart-muscle, characteristic symptoms.

Let us suppose that the valvular lesion is that of mitral regurgitation with failure of compensation. The first and one of the most prominent symptoms is shortness of breath on exertion; the lips and ears do not possess their normal red hue, but are a little bluish; and if the congestion of the auricle and pulmonary veins is great, bronchitis may be constant or attacks of hæmoptysis may develop. Palpation of the heart will also be complained of; and if the patient has developed the lesions in early life, the finger-tips are apt to be clubbed. If the rupture or failure of compensation is more complete, all these symptoms become more marked, and the shortness of breath, even when lying down, becomes most distressing; indeed, the patient may be comfortable only when sitting up. Dropsy of the lower extremities now comes on; the liver becomes enlarged from portal congestion, and this results in disordering gastric digestion by the catarrh of the stomach which ensues, while the urine becomes albuminous, not necessarily from any true renal lesion, but as the result of engorgement of the kidneys with blood.

The general symptoms of mitral obstruction are identical with those just described. For the differential physical signs see page 302.

The general symptoms of aortic obstruction are also much like those described as resulting from mitral regurgitation, but in addition there are apt to be present, early in the process of failing compensation, some lightness of the head, dizziness or vertigo, or faintness, owing to a deficient blood-supply to the brain. Very commonly, too, it will be found that in association with the aortic stenosis there also exists mitral regurgitation, which speedily produces in its turn well-marked pulmonary symptoms. Dropsy is very rarely seen in patients with aortic stenosis. On the contrary, they present, as a rule, the lean and poorly nourished appearance so often found in the adult, well advanced in years, who has atheromatous tendencies in the bloodvessels.

The association of ruptured compensation with aortic regurgitation presents more typical general systemic symptoms than any of the ordinary valvular lesions of the heart. In addition to headache, vertigo, and a tendency to syncope associated with palpitation and a sense of cardiac oppression, we often have a great deal of cardiac pain, of a dull, aching character in rare instances, but more often intensely sharp and lancinating, often darting down the left arm, particularly at night. The dyspnœa is often extreme, the patient suffering from terrible attacks of shortness of breath and often sitting day and night in a chair with his head resting on the back of a chair placed in front of him. As time goes on the constant struggling for breath exhausts him, and he falls asleep, only to wake in a few moments gasping for air. Long before any of these grave symptoms arise we may, however, find a number of interesting signs of this heart-lesion, chief among which is the "water-hammer" or "trip-hammer" or "Corrigan pulse," the throbbing arteries, and capillary pulsation in the skin and mucous membranes is to be seen. The last is best developed by drawing the thumb-nail sharply across the forehead, thereby causing a red mark, which can be seen paling and flushing with each beat of the heart (Quincke's sign), or by pressing a glass slide on the inner part of the lower lip, when the same capillary pulsation will be found. Ophthalmoscopic examination will often reveal similar pulsation of the retinal arteries.

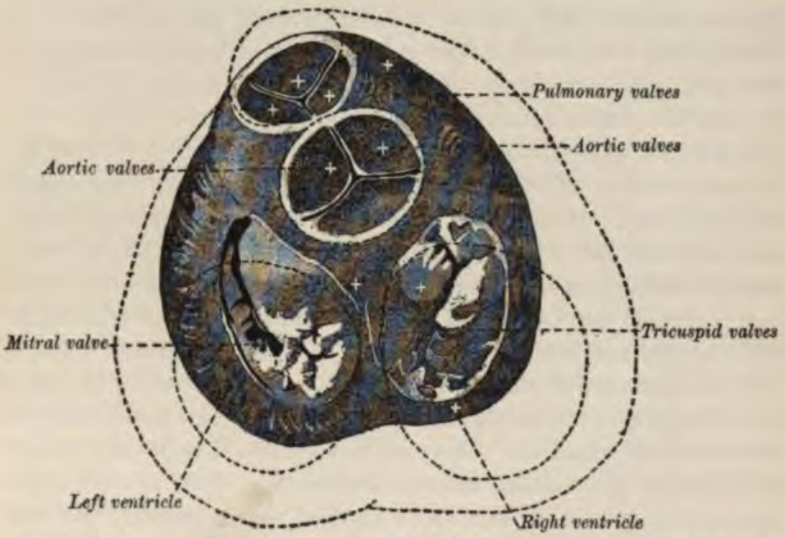
Beyond valvular lesions, producing heart-symptoms, we have a number of other causes which seriously disturb the action of the heart and the general circulatory condition. The first of these is dilatation of the heart, independent of associated valvular disease. Let us suppose that a man presents himself with a history of shortness of breath on exertion, so great that his activities are greatly reduced and his usefulness impaired. He gives a history that he was well until he made some extraordinary exertion. Since that time his symptoms of heart-failure have been marked. He may perhaps have attacks of syncope. Examination of his heart reveals on inspection a diffuse thrill in the region of the apex; but this thrill is too feeble to be felt, though well marked to the eye if his chest is thin. Percussion shows that the area of cardiac dulness is increased vertically and laterally, and auscultation will discover feeble heart-sounds. If the dilatation of the muscular portion of the heart is associated with dilatation of the orifices, a murmur may be present, most commonly that of mitral regurgitation, without there being in association any actual disease of the mitral valves. Sometimes tricuspid regurgitation is also found. The first sound, before it becomes very feeble, may be short and flapping like the ordinary second sound. Marked arhythmia of the heart is often present.

The influence of severe strain in producing cardiac disease deserves careful study on the part of the physician. The study can be divided into three parts: The condition of the heart immediately after acute overstrain, the condition after chronic overstrain, and the final condition often met with months or years after the occurrence of the strain.

It is now well known that immediately after severe muscular effort an examination of the heart will often reveal in entirely healthy persons a distinct increase in the area of cardiac dulness and, not infrequently, a murmur which disappears with rest. The increase in the area of dulness is, of course, due to more or less cardiac dilatation and the murmur to the same causes, since the dilatation results in stretching of the circular muscular fibres governing the mitral or tricuspid orifices, so that even if the valves be healthy they cannot close the orifice. (See Fig. 130.) In some cases this action seems to be in the nature of a safety-valve, in others a sign that the heart has been unduly strained. In the first class the murmur disappears at once or very shortly after the exertion ceases; in the other case it persists until after a long period of rest, when the heart has had a chance to recuperate and regain its normal tone. Persons having the latter condition ought to be advised against such forms of exercise, particularly if they are old in actual years or prematurely aged.

But the physician who regards physical strain as being the only kind of strain which is apt to be productive of grave cardiac damage is much astray. It is quite true that sudden cardiac failure due to great nervous or mental shock is not common, most of such attacks being due to vasomotor relaxation and being quickly recovered from. It is remarkable how often we meet with hearts which are distinctly damaged by prolonged nervous worry. I have met with this not infrequently in men who have been most successful in business or in their professional life, men whose success has been largely due to great persistency of effort, who have had great mental strains in carrying through some business deal, and have simultaneously used a good deal of physical force in long journeys or busy days on foot at the same time. Not only do such cases develop the various cardiac neuroses, but not rarely such lives seem to bring on the development of actual organic and degenerative changes in the heart muscle and bloodvessels. That these changes are the result of nerve strain more than infection or exposure is shown by the frequency of these states in the officers of great corporations and their infrequency in the employes who perform the manual labor. There can be no doubt that one cause of this lies in the great increase in the heart action required by mental effort, accompanied as it is by nervous waste without the compensation of long periods of rest, of outdoor exercise, and fresh air. Recent studies with instruments of precision have shown that increased mental activity causes circulatory activity to a greater degree than is generally thought. Such persons run at a high tension for years, develop a state of chronic vascular spasm and high arterial tension, and break down when the heart finds itself

FIG. 130.



The dotted lines show the dimensions to which the heart and its orifices increase in diastole, and in pathological dilatation this may be permanent. (Modified from His and Spalteholz.)

tired out with the stress laid upon it. These patients, too, when failure of energy first begins to creep upon them, use their will power to drive them to greater effort, and often very unwisely use stimulants to act as a spur to greater effort.

Again, we have hypertrophy of the heart occurring in persons without valvular lesions, sometimes as the result of excessive and severe toil. It is seen most commonly by the author in medical students, who, during their holidays, devote their time to severe athletic sports, or to much manual labor, and who, on leading sedentary lives in the winter, develop irregular cardiac action, palpitation, and some shortness of breath. Examination of the præcordium in such cases shows a forcible impulse of the apex of the heart against the chest-wall, some bulging of the chest-wall if the hypertrophy be very great, and no murmurs, but in their place heart-sounds very much louder than normal. Palpation shows the apex-beat to be lower than normal, and on percussion an increase in the area of cardiac dulness is also found.

Again, let us suppose that a patient presents himself with the statement that he has attacks in which he suffers from a very rapidly beating heart. His skin is alternately red and pale, and sweats without cause, but a careful examination of the heart fails to reveal any murmurs or organic abnormality. There are considerable shortness of breath on exertion and marked palpitation and arhythmia. Such a case may be suffering from a condition in which there is some deficient action of the pneumogastric nerve, whereby the heart is not properly controlled, or the irregular cardiac action may be due to sudden vasomotor relaxations, which by dilating the blood-paths reduce the normal arterial resistance. This is a condition seen in association with some neuroses and very commonly met with in persons who use tobacco to excess. The symptoms of the so-called "tobacco-heart" are indeed chiefly those of arhythmia due to pneumogastric disorder.

Closely associated with these patients is that class who suffer from prolonged feebleness of the heart after an attack of true influenzal infection, a feebleness which may last for many years. In many of these patients it is most difficult to determine how much the feebleness of the heart depends upon real changes in its muscular fibres and how much upon lack of proper nerve supply to this organ. Where there is a history of previous cardiac difficulty there can be little doubt that the poison of this disease magnifies it and produces far too often a permanent increase in the disease. In other instances where the heart is primarily healthy the condition is usually a fleeting one, although it is not to be forgotten that undue muscular activity on the part of the patient soon after or during an attack of influenza may be provocative of permanent cardiac incompetency.

What the exact changes in the heart are under these circumstances has not, so far as I know, been determined, but during life we certainly meet with them clinically. Such patients often suffer from faintness and precordial distress on exertion, and a physical examination usually reveals a feeble and distant first sound, and a second sound lacking in tone because of lack of force in the systole of the heart. These patients also have œdema of the lower extremities, sometimes a more or less decided trace of albumin in the urine, but no casts unless there be associated renal lesions, the albuminuria being due to stasis in the kidney. They are often pallid and relaxed, and really need rest and hydro-therapeutic measures more than the internal use of drugs. Such patients too, particularly if they are advanced in years, not rarely progress rapidly into a gradually increasing circulatory feebleness and so to death.

There is another heart condition closely allied to that just described, at least in one sense, namely, the feeble heart of tuberculosis. Too little attention is paid to this organ in this disease, probably because the mind is centred upon the lungs.

Rarely because of irritation of the vagus nerves or centres a state of bradycardia develops, in which the heart beats very slowly, perhaps only thirty or even as slowly as twelve times a minute. Bradycardia, or great slowness of the heart, may be due to a neurosis of the vagi, and also occurs in some infectious diseases. It is also seen in jaundice.

One of the most common causes of tachycardia, or rapid heart, is exophthalmic goitre, in which condition we have not only exophthalmus and enlargement of the thyroid gland, but, in addition to the tachycardia, a marked thrill over the carotid arteries, in which vessels a purring murmur of considerable intensity can also be heard. The patient often suffers from considerable nervous excitement or mental depression. It is an interesting fact that in this disease the electrical resistance of the body is often diminished.

An exceedingly irregular arhythmical action of the heart coming on in the course of an acute infectious disease, or in any state productive of sepsis, points to the possibility of the patient having an embolism or thrombosis of one of the coronary arteries. If the vessel is suddenly plugged, death occurs; but if the process is gradual, an anæmic necrosis or white infarct is produced.

Before discussing the signs of so-called fatty heart we must decide what is meant by this term. True fatty heart—that is, the condition of the heart in which this organ has undergone true fatty degeneration—has no pathognomonic signs, so far as the heart itself is concerned. In these instances we base our diagnosis upon the presence of fatty degeneration of the more superficial organs, such as the arcus senilis in the eye,[1] the presence of atheromatous bloodvessels, the feeble heart-sounds at all times, and the evident feebleness of the heart on exertion. The history of poisoning by any one of the poisons causing fatty degeneration is also to be sought after in some cases. Marked fatty degeneration is often present in cases of pernicious anæmia. It is not possible to make a differential diagnosis from the physical signs between fatty and fibroid heart.

Another state quite distinct from true fatty heart, but with somewhat similar symptoms, is seen in cases in which an excessive amount of fat has been deposited round the heart as well as in or around the other organs of the body. Here there is little or nothing the matter with the heart-muscle, except that it is overloaded with a weight of fat.

When a man shows signs of general degenerative changes, has a feeble heart, some dyspnœa, and perhaps some œdema of the lower extremities, we may conclude that he has, unless valvular disease is discovered, degenerative myocarditis. Valvular disease may, of course, be found associated with the myocardial lesion. Such cases make up the greater number of sudden deaths, called popularly "death by sudden cardiac failure."

Great feebleness of the heart and of the general system, loss of flesh (or sometimes maintenance of weight), and pigmentation of the skin and buccal mucous membranes point strongly to Addison's disease (see chapter on Skin).

Sudden attacks of cardiac feebleness sometimes come on as cardiac crises in glosso-labio-pharyngeal paralysis and in locomotor ataxia.

Finally, let us suppose that a young child is seen who is, and has been since birth, more or less cyanotic. In all probability such a child is the subject of congenital malformation of the heart. The following rules, laid down by Hochsinger, may be used for making the diagnosis:

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[1] Ophthalmologists and many clinicians deny that arcus senilis has any significance of this character. (See chapter on Eye.)

1. In childhood loud, rough, musical heart-murmurs, with normal or slight increase in the heart-dulness, occur only in congenital heart-disease. The acquired defects with loud heart-murmurs in young children are almost always associated with great increase in the heart-dulness.

2. In young children heart-murmurs, with great increase in the cardiac dulness and feeble apex-beat, suggest congenital changes. The increased dulness is chiefly of the right heart, whereas the left is only slightly altered. On the other hand, in the acquired endocarditis in children, the left heart is chiefly affected and the apex-beat is visible; the dilatation of the right heart comes late and does not materially change the increased strength of the apex-beat.

3. The entire absence of murmurs at the apex, with their evident presence in the region of the auricles and over the pulmonary orifice, is always an important element in differential diagnosis, and points rather to septum defect or pulmonary stenosis than to endocarditis.

4. An abnormally weak second pulmonic sound associated with a distinct systolic murmur is a symptom which, in early childhood, is to be explained only by the assumption of a congenital pulmonary stenosis, and possesses, therefore, an importance from a point of differential diagnosis which is not to be underestimated.

5. Absence of a palpable thrill, despite loud murmurs which are heard over the whole præcordial region, is rare, except with congenital defects in the septum, and it speaks therefore against an acquired cardiac affection.

6. Loud, especially vibratory, systolic murmurs, with the point of maximum intensity over the upper third of the sternum, associated with a lack of marked symptoms of hypertrophy of the left ventricle, are very important for the diagnosis of a persistence of the ductus Botalli (ductus arteriosus), and cannot be explained by the assumption of an endocarditis of the aortic valve.

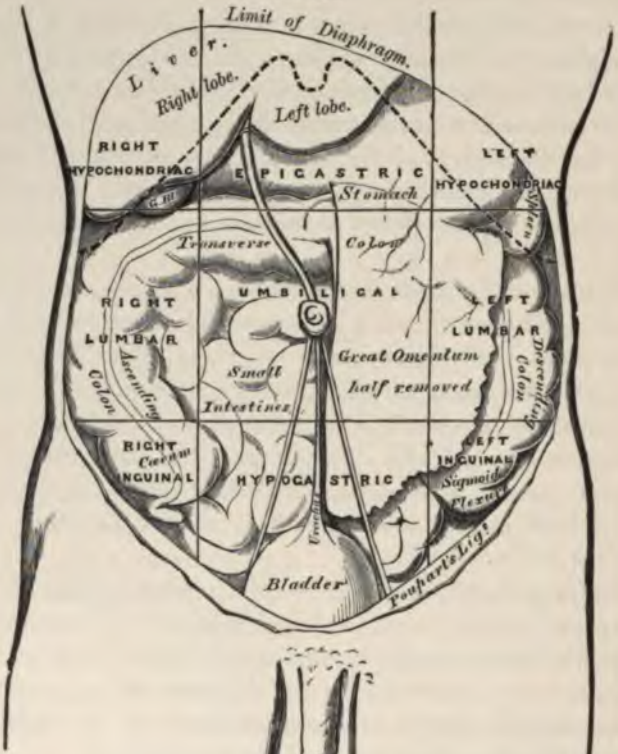
CHAPTER VIII.

THE ABDOMEN AND THE ABDOMINAL VISCERA.

The surface of the abdomen—Changes in the appearance and shape of the abdominal wall—The signs and symptoms of disease of the abdominal organs.

THE condition of the abdominal surface and abdominal contents is best studied by means of inspection, palpation, percussion, and auscultation. For the purposes of inspection the surface of the abdomen has been arbitrarily divided by diagnosticians into a num-

FIG. 131.



The regions of the abdomen and their contents. (GRAY.) (Edge of costal cartilages in dotted outline.)

ber of spaces, which are best shown in the accompanying figure (Fig. 144), and which get their names from the regions in which they are located, or from the organ immediately underneath the abdominal wall. By means of these arbitrary outlines we can readily describe the exact spot in which a physical sign or symptom is found.[1]

The following table, from Gray's *Anatomy*, clearly shows the viscera to be found under each of the areas named:

Right Hypochondriac.	*Epigastric Region.*	*Left Hypochondriac.*
The right lobe of the liver and the gall-bladder, hepatic flexure of the colon, and part of the right kidney.	The pyloric end of the stomach, left lobe of the liver, and lobulus Spigelii, the pancreas, the duodenum, parts of the kidneys and the suprarenal capsules.	The splenic end of the stomach, the spleen and extremity of the pancreas, the splenic flexure of the colon, and part of the left kidney.
Right Lumbar.	*Umbilical Region.*	*Left Lumbar.*
Ascending colon, part of the right kidney, and some convolutions of the small intestine.	The transverse colon, part of the great omentum and mesentery, transverse part of the duodenum, and some convolutions of the jejunum and ileum, part of both kidneys.	Descending colon, part of the omentum, part of the left kidney, and some convolutions of the small intestine.
Right Inguinal (*Iliac*).	*Hypogastric Region.*	*Left Inguinal* (*Iliac*).
The cæcum, appendix cæci.	Convolutions of the small intestine, the bladder in children, and in adults if distended, and the uterus during pregnancy.	Sigmoid flexure of the colon.

INSPECTION. On inspecting the abdominal surface the physician should look for eruptions which may indicate some general disease, as typhoid fever; for localized swelling, which may be due to hernia; for striæ, indicating that the skin has been stretched by excessive fat, by great swelling from ascites, or by pregnancy. He should also in a case of suspected early pregnancy look for the dark line in the linea alba. If the umbilicus is protruding and tense it may indicate distention due to grave abdominal disease, or it may be infiltrated by a morbid growth which has been primarily hepatic. If it be a secondary growth the navel will be "moored fast." Sometimes it is much swollen from chafing and eczema. Umbilical hernia may be found.

The general abdominal wall is protruded and retracted in normal respiration in both sexes, but more markedly so in males. It is pushed outward or protruded by many perfectly normal causes, such as an unusual amount of fat in the omentum, pregnancy, and an accumulation of liquid and food in the stomach after a heavy meal. It is also convex to an abnormal degree in cases in which ascites is present, when the stomach and bowels are over-distended with gas (tympanites), and when any of the organs found in the peritoneal cavity are the seat of swellings or tumors of large size. (See Figs. 146, 147.) In children a protruding pot-belly, "the frog-belly" of the French, is seen in cases of scrofula or tuberculosis of the mesenteric glands, and in those cases which suffer from chronic gastro-intestinal catarrh. It is claimed in a recently published paper by a French clinician that the intestinal canal is not only dilated, but of greater length than is normal in these cases. If, on the other hand, the belly-wall is retracted, concave, or "scaphoid," as it is sometimes called, we look for the cause in abstinence from food, or remember the possibility that excessive vomiting or purging may have emptied the gastro-intestinal tract of its usual contents. Thus excessive summer diarrhœa may produce such a result. We also find a retracted belly-wall in nearly all cases of advanced wasting diseases, such as carcinoma, or tuberculosis of the lungs; and if the retraction is associated with muscular rigidity of the belly-wall and pain, we suspect the early stages of peritonitis or the presence of some acutely painful affection, such as renal or hepatic colic or lead colic. Marked concavity and retraction of the belly-wall are also seen sometimes in cases of tubercular meningitis.

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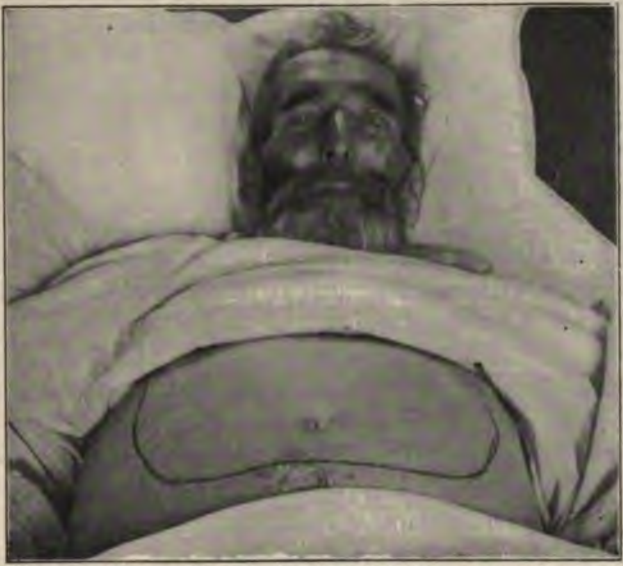
[1] For changes in the skin of the abdomen, see chapter on the Skin.

Sometimes in thin patients peristaltic waves are to be seen traversing the abdominal surface as the result of violent movements of the bowels. These waves are commonly seen in cases of intestinal obstruction, and in neurotic persons with muco-membranous enteritis, or, if in the epigastrium, may be due to a dilated stomach. If the waves are from below upward and in the right side, they are probably arising in the ascending colon; if from above downward and in the left side, in the descending colon and sigmoid flexure. Again, gastric waves pass from left to right, while those in the transverse colon pass from right to left.

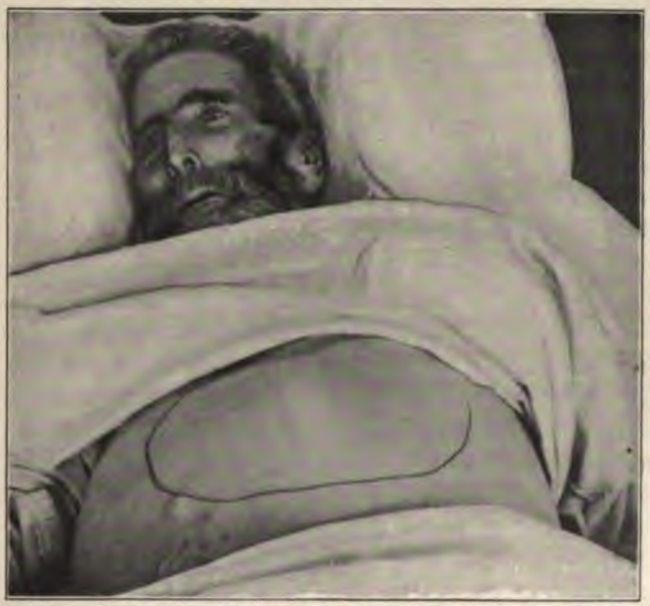
The abdomen is distended very greatly by gas in many cases of peritonitis, typhoid fever, and in persons suffering from flatulent colic. If this be the cause of the distention, percussion of the anterior belly-wall when the patient is lying on the back will give a tympanitic note. We separate, diagnostically, the swollen abdo-

FIG. 132.



Enormous ascites. The area inside the line is that which was resonant on percussion (gut tympany). On either side and below the line there was absolute flatness due to the presence of fluid. (From the author's wards.)

FIG. 133.



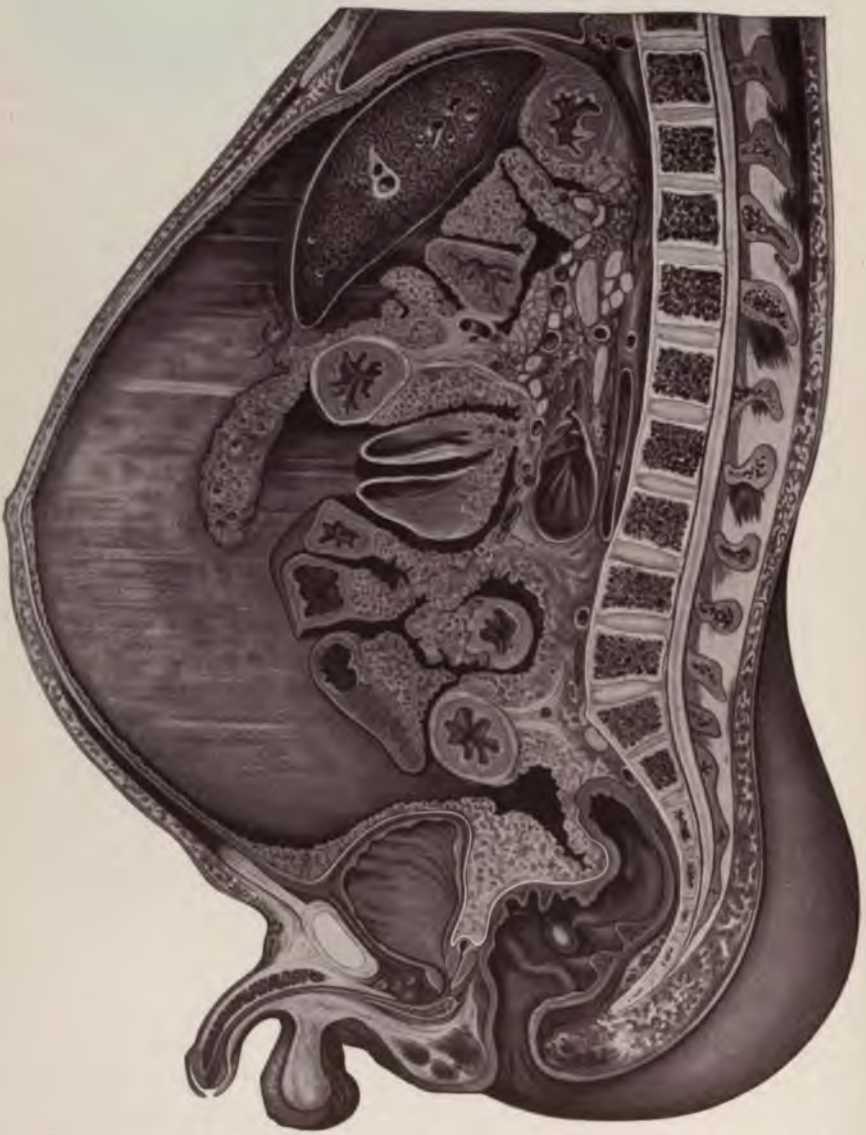
Same as figure above. When the patient was turned on the side the fluid flowed in that direction, and so the area of resonance was altered, the area of flatness on the right increasing and on the left diminishing.

men due to wind from that due to ascites by the fact that in the latter condition the epigastrium is moderately flat when the patient is lying down, while when tympanites is present it is more protruding. Again, in ascites the greatest bulging is generally to be found in the flanks, or, if the patient sits or stands erect, the hypogastric region bulges from the change in the position of the fluid. If the swelling be due to a moderate-sized ovarian cyst, this variation in form will not occur, as the cyst is not readily movable. If the ovarian tumor be large, the differential diagnosis may be most difficult and almost impossible, except by the history or by examining the liquid withdrawn by tapping.

In cases of ascites due to free liquid in the abdominal cavity percussion will elicit flatness over the flanks and resonance where the intestines containing gas are floated up against the anterior belly-wall above the effusion. Sometimes, however, if the large intestine be empty of fecal matter, percussion in the flank behind the midaxillary line will reveal tympany, because the peritoneum walls off the liquid from the posterior surface of the bowel. If the patient is turned slightly to one side, the area of flatness on percussion is altered, as is shown in Fig. 133. Palpation will also reveal fluctuation in ascites, but none in tympanitic distention. To develop this fluctuation, the patient is placed on his back and the finger-tips of the left hand of the physician are placed against the skin of the flank. With the finger-tips of the right hand the opposite flank of the patient is struck a blow as in performing ordinary percussion, when the impulse, if fluid is present, will be transmitted to the fingers of the left hand. To prevent a transmission of the impulse through the abdominal wall, an assistant may press with the edge of his hand over the linea alba. (Fig. 134.) As the result of gradually increasing intra-abdominal pressure the floating ribs become pushed outward, the apex-beat of the heart is often displaced upward and outward, and the umbilicus becomes protruded instead of retracted. The skin of the belly-wall becomes thin and shining, and the recti muscles may become separated. After tapping in such cases the peristaltic movements of the bowels can be readily felt through the intervening skin.

Having decided that the distention is due to an accumulation of free fluid in the abdomen, it remains for the physician to determine what the cause of the ascites may be. Its most frequent cause is atrophic cirrhosis of the liver, which results in engorgement of the

PLATE XV.

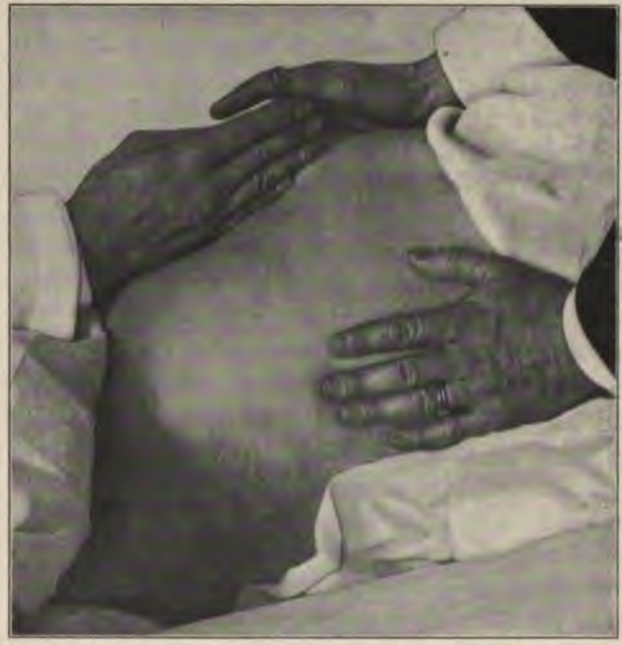


Median Vertical Section of Body of a Boy of 17, who Died of Colloid Cancer of the Peritoneum and Iliac Flexure.

The gelatinous masses represent the growth and the ascitic fluid is readily seen. (Ponfick's Atlas.)

abdominal vessels with secondary transudation of fluid (Fig. 134). If it be not due to cirrhosis, it may arise from an abdominal tumor, which by pressing on large vessels results in an effusion of liquid through their walls (Plate XV.), or be caused by tubercular peritonitis, by obstruction of the thoracic duct, by valvular disease of the heart causing an obstruction to the flow of blood in the vena cava, or, finally, by chronic parenchymatous nephritis. If the last cause be present, there will usually be some œdema of the lower extremities or general anæmia with dyspnœa and albuminuria. For the typical symptoms and physical signs of these various affections the reader is referred to those parts of this book in which they are discussed (see Index).

FIG. 134



Showing method of determining the presence of fluid in abdomen by transmitted fluctuation. The hand of an assistant is placed on edge on the middle line, to prevent transmission of impulse by the belly-wall. The right hand then taps the flank, and if fluid is present the impulse is felt by the left hand on the other side.

There is an additional source of information to be utilized as to the cause of the ascites, namely, the character of the effusion. If the fluid withdrawn on aspiration has a specific gravity of

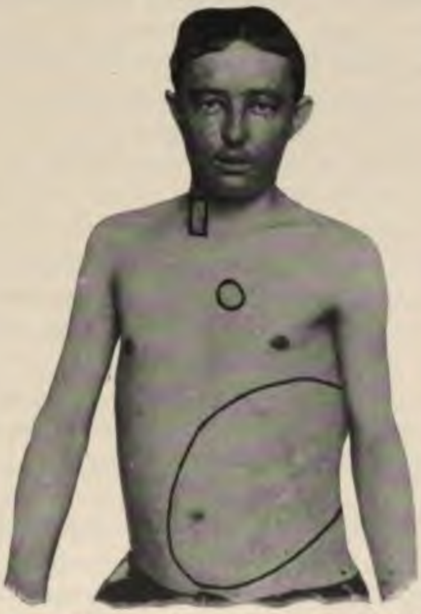
1.008 and contains but a trace of albumin (about 0.97 per cent.), it is probably due to hepatic cirrhosis, whereas if due to the pressure of a tumor the specific gravity is usually about 1.012 and the albumin nearly 2 per cent. Such a specific gravity and proportion of albumin may also result when the ascites is due to heart disease or to pressure on the thoracic duct. When the effusion is the result of Bright's disease the specific gravity is apt to be only 1.006 and the proportion of albumin only a trace. In cases in which the ascites arises from some disease directly affecting the cells lining the peritoneal cavity, as carcinoma of the peritoneum or of the abdominal viscera, or tubercular peritonitis with or without pus, the specific gravity is much higher than just stated, namely, from 1.018 to 1.027, as a rule, and the proportion of albumin ranges from 3.80 in the case of growths to 5.76 in the case of tubercle and 7.10 when there is pus. To express it otherwise, the percentage of albumin is in direct proportion to the inflammatory process, and an effusion due to hydræmia would therefore contain only a trace of albumin. Further than this, it is asserted by Pohl and Rosenbach that the effusions due to venous engorgement, heart disease, and renal lesions can be separated from those due to disease in the peritoneum affecting this membrane directly by a test following the administration of iodide of potassium. When this drug is given to the first class of cases it speedily appears in the effused fluid; but should effusion be due to the organic diseases of the peritoneum which have been named, it will not appear. The fluid to be tested is placed in a test-tube and some nitric acid and chloroform added, when if iodine is present its characteristic color will appear. Should the cause of the ascites be a ruptured ovarian cyst the diagnosis of its cause, except from the history of a previous localized swelling, is exceedingly difficult. The fluid under these circumstances is usually of a specific gravity of 1.026, but its specific gravity may be much lower. It is asserted that in the instances in which the specific gravity of the fluid is very low the swelling is due to a cyst of the broad ligament.

Rotmann asserts that in all serous effusions, even in non-diabetic patients, there is present a trace of sugar; whereas in all seropurulent effusions sugar is absent.

Very often in cases of ascites, particularly when this condition arises from hepatic cirrhosis, there is developed on the anterior belly-wall a more or less well-defined bunch of veins, which is sometimes called the *caput Medusæ*, as the result of an attempt at collateral circulation, to compensate for the obstructed flow caused by the changes in the liver. Sometimes a mediastinal growth will cause a somewhat similar development. When the obstruction is lower down than the liver the superficial veins of the lower part of the abdomen (hypogastrium) will be found distended.

Localized bulging of the abdominal walls, chiefly on the right side, is found in cases in which the liver is enlarged by hypertrophic cirrhosis, or by cancer or other morbid growth, such as gumma or sarcoma, and by abscess. The swelling, if its origin be in the liver, will arise under the floating ribs on the right side, and will extend downward and forward toward the umbilical area. If the enlargement be great, it will extend far below the umbilicus and across the umbilical area to the opposite side of the abdomen, as in Fig. 145. In enlargement of the spleen similar signs, springing from the floating ribs well over to the left side, may be developed (see Fig. 135), and large cystic kidney on either side may cause abdominal

FIG. 135.



A case of splenic anæmia with great enlargement of the spleen, as shown in the large outlined area. The smaller outlines indicate the areas of anæmic murmurs near the base of the heart and in the carotid artery. (From the author's wards in the Jefferson Medical College Hospital.)

bulging, particularly if the kidneys be floating. (See Floating Kidney and Spleen.)

Marked swelling of the epigastrium indicates distention of the stomach by gas or food, or that this organ is the seat of morbid growth. Sometimes a similar distention results from enlargement of the posterior mediastinal and retro-peritoneal glands. Again, distention of the epigastrium is apt to be caused by enlargement of the left lobe of the liver. In ovarian tumors the growth often gradually distends the entire belly equally; but, as already stated, the history is usually that of swelling, low down, and of its being chiefly unilateral at first.

It should be remembered that the discovery of a pyriform swelling in the hypogastrium may possibly be due to a pregnant uterus, or to retention of urine, with consequent distention of the bladder.

Cases of dilatation of the stomach often show very great bulging of the umbilical area of the abdominal wall when that viscus is distended by liquid and gas. (See Percussion, in this chapter.)

In inspecting abdominal swellings the physician should watch to see if they move up and down with respiration. If they do, they are probably connected with the diaphragm and depend upon disease of the liver and spleen, as tumors of the pancreas, stomach, and kidney are usually not attached to the diaphragm, and therefore generally do not move. Inspection of the abdominal wall will also show possible venereal infection if the glands in the groin are enlarged, or if in suppurating they have left puckered scars. If silvery lines extend across the belly, they may indicate pregnancy past or present, or any state of the abdominal tissues causing great stretching of the skin. Great bulging in the neighborhood of the umbilicus will naturally suggest umbilical hernia, and swelling in the groin, adenitis, inguinal hernia, or perhaps an appendicular abscess.

PALPATION AND PERCUSSION. More important than any other external method of studying the condition of the abdominal contents is the use of gentle *palpation*, the fingers being gradually worked down into the abdominal cavity in such a way as not to cause pain or excite the muscles of the abdominal wall to resistance. The hand should always be carefully warmed before palpation is attempted, and the object of the examiner is to discover, first, the hardness or resistance to pressure; secondly, the consistency and form of the organs which he can touch; and, thirdly, whether any swellings which he feels are movable, bound down and immovable, pulsating, soft or hard, nodular or smooth. The patient whose abdomen is to be palpated must be placed flat on his back, with the knees draw up to relax the abdominal muscles, the head and neck should be raised, and, if possible, the attention of the patient should be diverted by conversation about some symptom which exists elsewhere than in the belly, while the examination is made, as in this way voluntary muscular resistance is removed to some extent. He should be made to breathe easily through his opened mouth; and if the belly-wall remains so rigid that a perfect examination is impossible, and yet the results of such an examination are very important, ether or chloroform should be given to relax the muscles. In other instances in which it seems inadvisable to give an anæsthetic, the patient may be placed in a bath as hot as he can bear. He should be entirely submerged up to the neck. The hot bath often relaxes the abdominal wall enough to aid diagnosis very greatly.

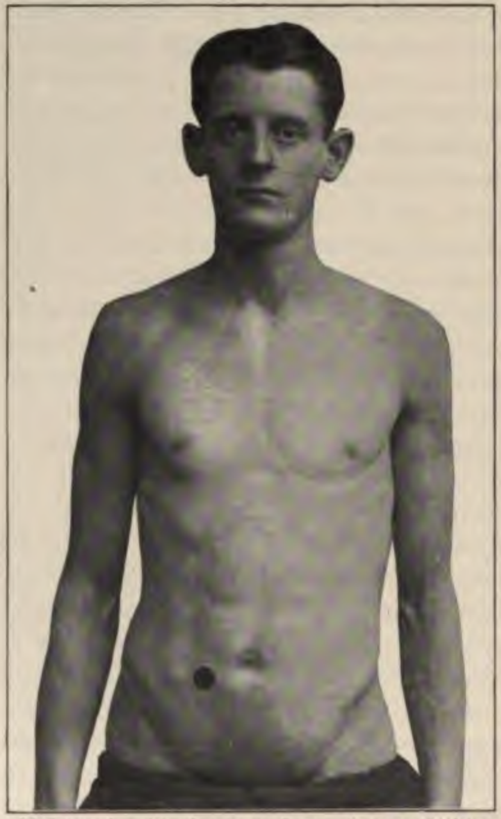
After the abdominal contents have been carefully examined, the patient being on his back, he should be placed first upon his left side and then upon his right, and the abdominal contents again palpated. This is particularly necessary when examining the belly for growths or when enlargement or displacement of the liver, spleen, or kidneys is suspected.

Great resistance of the rigid abdominal muscles is found whenever peritonitis is present in an acute form, in some cases of renal and hepatic colic, and more commonly in lead colic and in hysteria. In peritonitis great tenderness to the slightest touch is also present. Another symptom of acute peritonitis, aside from the exquisite tenderness of the abdomen, the drawn lip, the thirst, and the distention or rigidity of the belly-wall, is pain of a severe character; unless it be septic peritonitis, when pain may be absent. There are also the drawing up of the limbs to relieve abdominal tension, obstinate constipation, moderate fever, and a very rapid, quick pulse. The tongue speedily becomes dry and parched, and collapse may soon ensue in severe cases. It is not to be forgotten that localized peritonitis may result from many causes, usually from disease of the appendix vermiformis or the genito-urinary tract in women, and that the local symptoms and lesions may be limited by a wall of lymph to a very small area of the abdominal cavity.

In appendicitis it may be general if the inflammation is widespread or localized, if the original area is limited or walled off from the rest of the abdominal cavity. If the rigidity be due to peritonitis, secondary to appendicitis, the following symptoms will point to an inflammation of the appendix as the cause:

There is usually a rapid pulse, which becomes more and more speedy as the gravity of the case progresses. Indeed, a very rapid pulse is a sign of great importance as indicative of the severity of the malady. There is marked tenderness at McBurney's point (Fig. 136) on pressure with the finger-tip. The pain may be referred to the epigastrium and the sigmoid flexure as well, but is not so severe on deep pressure in these parts. There is usually an increased leucocytosis (see chapter on Blood), an anxious face, and sometimes very great pain. (See chapter on Pain.) The febrile

FIG. 136.



McBurney's point of great tenderness in appendicitis.

movement is usually moderate, and it may be absent. It is to be constantly borne in mind that the early stages of typhoid fever, often so closely simulate appendicitis of the subacute type, that a differential diagnosis can only be reached after a most careful study of the case.

It must be remembered, however, that the anterior abdominal wall, particularly that of nervous persons, is often very sensitive or "ticklish," and the mere exposure of the skin to the air of the room, coupled with the fear of examination, may cause great rigidity of the belly-wall without there being any abnormal condition present. This can be generally overcome by gentleness in palpation and by resting the palm of the hand on the belly and partly flexing the fingers, rather than by attempting to insert the finger-tips between the abdominal muscles. The writer has seen a case of rhythmical hysterical spasm of the recti muscles in a male, which at first gave the sensation of an enormous diffuse pulsating aneurism of the abdominal aorta.

Let us suppose that, on placing the hand upon the epigastrium and the upper part of the umbilical area, we find a swelling. In the first place, we must decide as to whether it is in the abdominal wall or in the abdominal cavity. If it is in the wall, it will be movable with the tissues of the wall and readily grasped by deep palpation; but if in the abdominal cavity, the abdominal wall may be made to move over it unless it be attached to the parietal peritoneum. Let us suppose it is in the wall of the abdomen, what can the swelling be? It may be a fatty tumor; in which case its surface will be dimpled and resistant, probably not painful, unless the part has been inflamed by rubbing or an injury, and it will not fluctuate. There will generally be a history that the person has exercised constant pressure on the part, as in leaning against a bench or table. Again, it may be an abscess; but aside from the rarity of this condition, we can exclude such a possibility by the absence of pain and fluctuation, and the history of a severe injury.

Very much more commonly a swelling in the epigastrium, or upper umbilical area, is due to an intra-abdominal cause. In adults the most common cause is probably a growth (generally a carcinoma) of the pyloric end of the stomach. In other instances it is due, particularly in children, to enlarged lymphatic glands, as in tubercular disease of the mesentery. This is also sometimes seen in adults. Sometimes by reason of tubercular peritonitis a nodular mass is not only felt in this area, but an abscess containing tubercular pus may be formed and become surrounded by walls formed by the gluing together of the organs by lymph. Carcinoma of the pancreas may also cause a swelling in this neighborhood, or a cyst of the pancreas may be present. Aneurism of the abdominal aorta is also not to be forgotten. Sometimes, too, a distended or carcinomatous gall-bladder may project into this area.

If the growth be gastric carcinoma, the patient will be in or past middle life (probably between the fortieth and seventieth years, although cases may occur as early as thirty years); will have a history of constantly increasing pain and discomfort in the stomach; there will have been much sour belching, and perhaps vomiting of coffee-ground-looking material; marked loss of flesh and some cachexia will be present. The disease occurs twice as frequently in men as in women. According to Welch's statistics, out of 1300 cases of gastric cancer, 791 were in the pylorus, 148 in the lesser curvature, 104 in the cardia, 68 in the posterior wall, and 61 involved the whole stomach. The remainder were in the fundus, the greater curvature, or the anterior wall. The growth, if in the pylorus, is usually freely movable, and for this reason can be readily felt, and then is often momentarily lost to palpation. Its position is apt to change with the posture of the patient and the presence or absence of food in the stomach. Pain is usually elicited on deep pressure, and, if the growth be large and at the pylorus, the symptoms of dilatation of the stomach may be present, because that viscus is dilated through obstruction of the pyloric opening, which results in retention of the gastric contents. Similar symptoms may, however, be produced by a deposit of lymph around a pyloric ulcer, which, being changed into fibrous tissue, causes great thickening of the gastric wall with matting of the omentum about it, closely simulating the mass produced by a malignant growth or simple hypertrophic pyloric stenosis. When cancer involves the cardiac area of the stomach, the organ, instead of becoming dilated, becomes greatly diminished in size, and feels like a narrow band in the neighborhood of the left floating ribs and epigastrium. The diagnosis will be aided by discovering that the capacity of the stomach is very small and acid absent.

The pressure of a tumor which can be palpated in the neighborhood usually occupied by the stomach does not, of necessity, indicate that this viscus is diseased. Not very infrequently an abnormal position of the colon or of other portions of the viscera may result in the physician's mistaking a growth in these parts for a gastric cancer. Even carcinomatous lymph-glands may lead to this error. It is in these cases that the testing of the stomach contents is most useful in making a differential diagnosis. Thus if the normal gastric acidity be present we can exclude gastric carcinoma; even if the growth be carcinoma of the duodenum, the gastric juice usually remains normal. Not only does the examination of the gastric contents aid us in this manner, but if a growth in the duodenum causes stricture, it may cause the duodenal contents to regurgitate into the stomach, so that we find pancreatic digestion going on there. Boas asserts that the presence of some fecal liquid brown fluid in the stomach indicates jejunal stricture.

In considering the differential diagnosis of carcinoma of the stomach and bowel, it is to be remembered that the former is far more common than the latter. Thus Heimann found that out of 20,054 cases dying of carcinoma in the hospitals of Prussia, 10,537 involved the digestive tract, and of these, 4288 affected the stomach, whereas only 20 involved the small intestine and 224 the large intestine.

Sometimes in cases of chronic gastric ulcer the area involved becomes so indurated as to be felt as a hard mass through the abdominal wall. In such instances the points which aid us in separating the condition from gastric cancer are the fact that the patient is young and usually a woman; that the vomiting occurs immediately after taking food, for in gastric cancer it is only seen in most cases several hours after food has been taken; that there is no sign of gastric obstruction; that there is an excess of hydrochloric acid in the gastric contents in cases of ulcer, and an absence of this acid in cases of cancer. It is, however, a noteworthy fact that if the cancer involves only the pylorus, the acid may be present, because this part of the stomach is not that which secretes acid; and it is also to be remembered that a single test of the gastric contents is not to be depended upon, but that several should be made. Any disease which produces atrophy of the gastric tubules may cause an absence of hydrochloric acid, as, for example, grave pulmonary tuberculosis and renal or cardiac disease. Even catarrhal jaundice may temporarily arrest its secretion, and as jaundice is often present in gastric carcinoma, because of pressure or hepatic involvement, this fact should not be forgotten. In other words, the absence of hydrochloric acid is not pathognomonic of cancer; neither is its presence a guarantee of the absence of the growth. The result of an examination for this acid simply gives us additional evidence in making a diagnosis. Finally, as a rule there is no cachexia in cases of gastric ulcer, though there may be anæmia. There is usually in cases of ulcer no great loss of weight unless the symptoms have been present a long time.

In cases of gastric ulcer great pain is often produced by deep or even superficial pressure over the epigastrium, and a painful spot can generally be found on the back, about the angle of the right scapula.

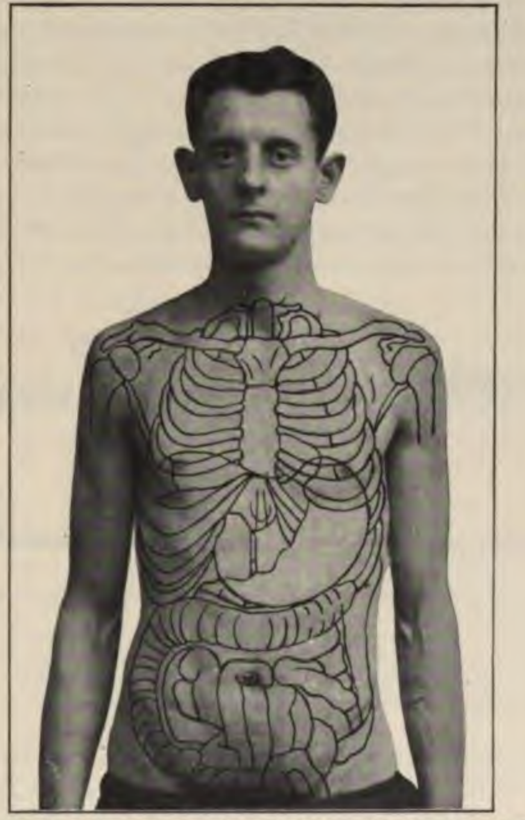
These painful spots are, however, as a rule quite localized, and the fact that they are very painful at one particular spot, and yet the surrounding parts are comparatively insensitive, points to ulcer as a cause. It is worth remembering, however, that duodenal ulcer may cause identical symptoms on palpation. Diffuse tenderness in this area is more apt to be due to colitis or even to appendicitis or sigmoiditis, which often results in pain in this area or in tenderness on percussion.

The presence of a resisting mass, deeply situated in the epigastrium, or the upper part of the umbilical area, and felt only on deep palpation, and then often indistinctly, should bring before the mind the possibility of the presence of carcinoma of the pancreas, a diagnosis which will be largely confirmed if cachexia be asserting itself, if there be great pain in this neighborhood, and if there are oily stools after fats are taken, as a result of the absence of pancreatic juice. Still further confirmation of this diagnosis will be present if diabetes mellitus be developed by the patient (pancreatic diabetes). Such a growth in the pancreas is usually scirrhous cancer, and may be primary or secondary. Segré found that of 627 cases of carcinoma of the upper abdominal organs cancer of the pancreas occurred in 127, but in only 12 of these primarily. Stiller asserts that the following symptoms are fairly sure signs of pancreatic cancer, namely, marked dyspepsia, rapid emaciation and cachexia, subnormal temperature, persistent and progressive jaundice without hepatic enlargement, but often with swelling of the gall-bladder from obstruction to its duct. These signs are, of course, only of value if the evidence of malignant growth elsewhere can be excluded.

Gastric dilatation results from obstruction of the pylorus or from inherent feebleness of the stomach-wall, and when it is present the entire upper part of the abdomen will be found distended, tense but yielding; and the history will show that the patient is attacked now and again by vomiting, during which a most extraordinary quantity of food and liquid, which has gradually accumulated, will be expelled.

We discover the condition of the stomach as to its size and shape by means of washing it clean with the stomach-tube and then filling it with a known quantity of water, which can be siphoned out and measured. Or instead we first wash out the stomach by means of a stomach-tube and then fill it with gas by giving the patient to drink, first, a half-glass of water with 1 drachm of sodium bicarbonate in it, and then another half-glass with 30 grains of tartaric acid dissolved in it, so that gas will distend the viscus. One is able with ease by means of percussion to outline the stomach over the area of which

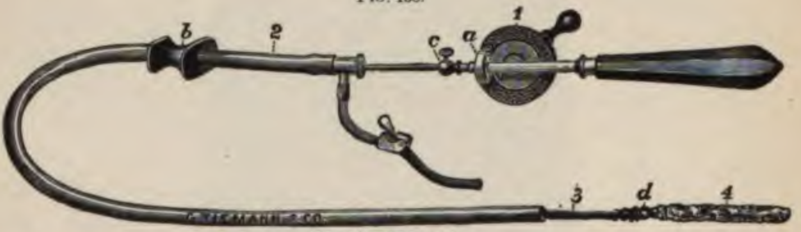
FIG. 137.



Outline of normal position and size of an adult stomach when distended with gas.

a high-pitched tympanitic note is heard. It is best to mark the edge of gastric resonance by means of a blue pencil, and thus map out the gastric area. Instead of this, we may distend the stomach with air by attaching a Davidson syringe to a stomach-tube or by using an atomizer bulb for the same pumping purposes. In other instances we can use the so-called gastrodiaphane of Einhorn, which consists of a small electric lamp, protected by strong glass, and attached to a rubber tube which contains the necessary wiring for the electric current, and which is swallowed just as is the ordinary stomach-tube. The stomach should first be thoroughly cleansed by lavage, then filled with pure water and the lamp swallowed. If the patient be moderately thin and the inspection is made in a dark room, the outline of the lighted stomach can be seen through the abdominal wall and some idea of its dimensions obtained. Normally, the greater part of the stomach will be found to the left of the middle line and about one to two inches above the umbilicus. (See Percussion.)

FIG. 138.



Türck's gyromele.

a represents a thin metal wheel, covered on one side by soft, unpolished rubber, which rotates on a smaller wheel by friction.

2 is the stationary outer tube held by the button or spool *b*. At the end of the tube, which reaches to the cardia only, is a bearing within the tube to make the cable run more smoothly.

3 is the cable, which is fastened to the revolving apparatus by the screw *c*.

4 is the sponge, which fits into a socket of the cable, *d*, and may be removed at will.

Another useful method of diagnosing dilatation of the stomach is by means of the "gyromele" of Türck, which in its improved form consists of four parts, namely: a revolving apparatus; a stationary outer tube; a cable covered with tight-fitting rubber tube; and a sponge-spiral attached to the cable.

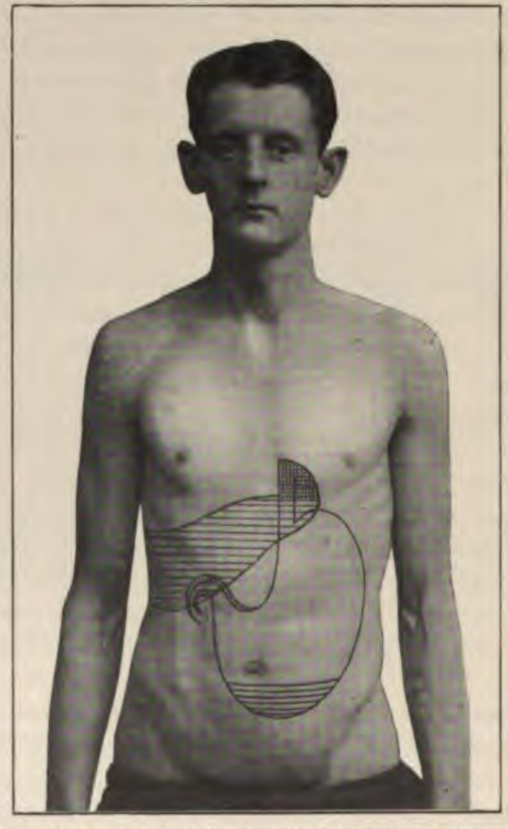
Türck says: "To show the outline of the greater curvature, a tube containing a cable with a sponge at its extremity is introduced into the stomach. An apparatus for producing revolutions is attached to the outer end of the cable. The cable is passed onward and it glides along the great curvature, plainly showing the outline of the stomach. Moving onward, the cable passes upward toward the pylorus, and then turns and passes along the lesser curvature. When rapid revolutions are produced the sponge and cable can be felt in their respective situations. To determine the distensibility of the stomach, cables of different degrees of flexibility are used. A very flexible cable (No. 1) is used first. It is introduced until it meets with resistance at the lesser curvature, and its length is noted. At the same time the revolving sponge is examined by palpation through the abdominal wall. A stiffer cable (No. 2) is then used, and pushed onward until it meets with resistance at the lesser curvature, and its length is noted. The lengths are compared, and their difference shows the degree of distensibility. The degree of distention also is found by palpation through the abdominal wall." (For the symptoms of gastric dilatation, see the chapter on Vomiting.)

Even before the stomach is artificially distended with gas percussion will give us valuable information in this connection, for, if obstruction of the pylorus exists, there will be found either a large area of gastric tympany through the accumulation of gas from fermentation, or, if no vomiting has taken place for some time, an equally great area of gastric dulness due to an accumulation of food and liquid. If there is a growth at the pylorus causing obstruction, there will be impairment of resonance wherever the pylorus may be situated. While this is a somewhat indefinite statement, it is to be remembered that a more definite one is liable to mislead the student, for even in health the position of the pylorus changes greatly when the stomach is empty or is filled with food. Thus, when empty the viscus hangs with the pylorus very low, but when it is filled the pylorus is raised. Fig. 135 shows the normal gastric area when the stomach is distended with gas. The following figures illustrate the extraordinary degree of descent of the stomach made in some cases of gastric dilatation in the adult. (Figs. 139, 140, 141, and 142.)

In some cases of dilatation of the stomach, or of gastroptosis, the use of "Glenard's belt sign" may be resorted to. This consists in standing behind the patient, placing the hands upon the lower part of the abdomen and lifting upward and backward, when if gastroptosis or enteroptosis is present relief from the sense of dragging may be felt.

Many of these cases of gastric dilatation are also associated with atrophy of the gastric tubules, or at least an absence of any secretion of normal gastric juice. The matters vomited or washed out of the stomach, are often devoid of hydrochloric acid, but loaded with an excess of lactic acid. Lactic acid is tested for as follows, the hydrochloric-acid test being given below: a few drops of neutral ferric chloride solution are mixed with one or two drops of pure carbolic acid, 10 c.c. of a 5 per cent. solution of carbolic acid, and water added until an amethyst hue develops. A few drops of the filtrate derived from the stomach-contents are now added, and if lactic acid or lactates are present the amethyst-blue will become yellow in color. This is a very delicate test for lactic acid.

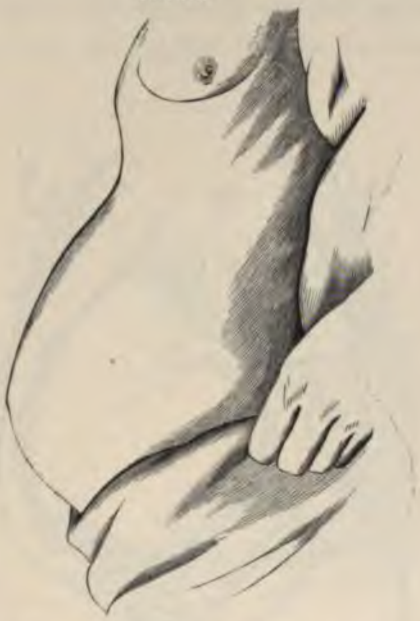
FIG. 139.



Showing the change in the position of the stomach in dilatation, and how difficult it is for this organ to empty itself of fluids.

There is no better place than the present to speak of the manner of testing the stomach-contents for hydrochloric acid. The patient is directed to take no food for at least twelve hours before presenting himself to the physician. On his arrival for examination he is given what is known as "Ewald's test-breakfast," which consists of an ordinary dry roll and a little over half a pint of water which has been warmed, and he is directed, after swallowing these materials, to wait for an hour. The stomach is now emptied by the introduction of the bulbed stomach-tube, and the gastric contents filtered. A few minims of a solution of phloroglucin and vanillin are next placed in a porcelain dish and a few drops of the gastric liquid are allowed to trickle down to the edge of the solution. The dish is gently heated over a spirit-lamp or Bunsen burner, and if hydrochloric acid is present there will appear a red tinge. This is an absolute proof of the presence of hydrochloric acid.

FIG. 140.



Profile view of the abdomen of a woman, aged sixty-five years, showing the tumor formed by the dilated stomach. (OSLER.)

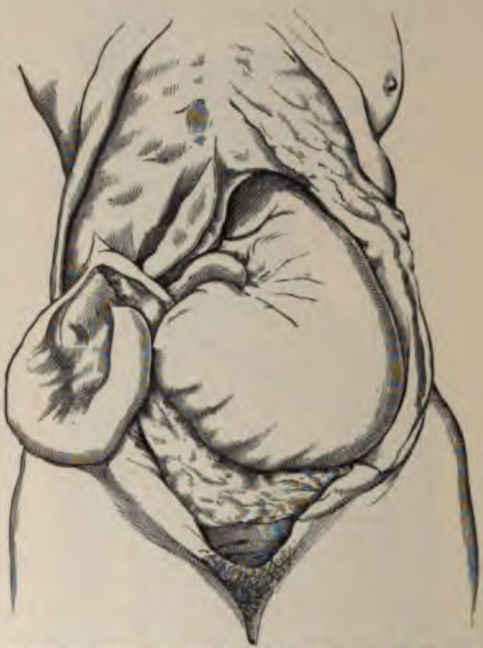
The solution of phloroglucin and vanillin is made as follows:

Phloroglucin	gr. xxx.
Vanillin	gr. xv.
Absolute alcohol	f℥j.

This solution is pale yellow in hue. It must be kept in dark bottles, as on exposure to the air and light it becomes brown and worthless.

If swelling of the abdomen is caused by tuberculosis of the abdominal organs the local signs may be quite varied, for such an infection may produce very varied conditions. Not rarely a mass, or masses, are felt in the abdomen which may be due to tuberculosis of the omentum, which causes a drawing up or puckering of the membrane, so that there can be felt a firm mass extending across the upper abdominal zone. Sometimes it can be felt to the right or left of the middle line. In other cases, but rarely, we find retracted and thickened coils of intestine which feel like tumor masses, and these are apt to be drawn against the spine, so that the belly is very scaphoid and empty. We also meet with cases in which large tubercular growths of the mesenteric glands occur. In the last group of cases and in those of the omental type, named above, there may be some ascites.

FIG. 141.

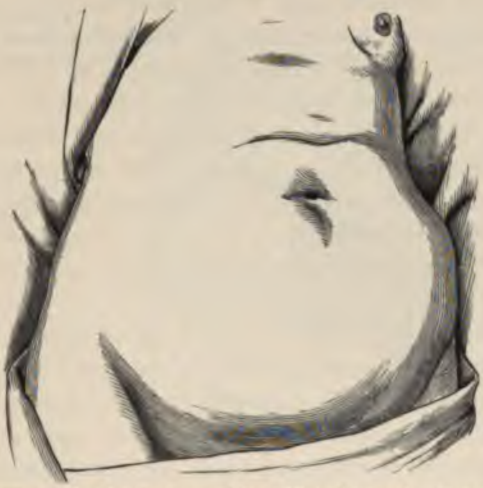


Showing the position and size of the stomach. (OSLER.)

Sometimes swelling in the upper abdominal zone is due to marked enlargement of the head of the pancreas owing to a malignant pancreatitis. This is apt to be associated with cholelithiasis.

The appearance of sudden swelling in the neighborhood of the pancreas, associated with intense pain, nausea, and vomiting, may be due either to acute hemorrhagic pancreatitis, to hemorrhagic infarction of the intestine, to intestinal obstruction, or to acute peritonitis resulting from perforation. (See chapter on Pain.) The last two are the more common. An exploratory operation is the only way of deciding the diagnosis positively, although the history of the patient may aid us in deciding the cause of the illness. Thus, if there is a history of gallstone colic, this may indicate that a stone has become impacted in the common duct near the papilla. Such an accident, if it dams back the secretion into the pancreas, causes hemorrhagic pancreatitis with fat necrosis. Sometimes, however, the symptoms of hemorrhagic pancreatitis are more prolonged, and life lasts for several weeks, local swelling, jaundice, and pain being present, with symptoms of suppuration. After death, not only is fat necrosis of the pancreas found to be present, but the general abdominal fat may be necrotic.

FIG. 142.

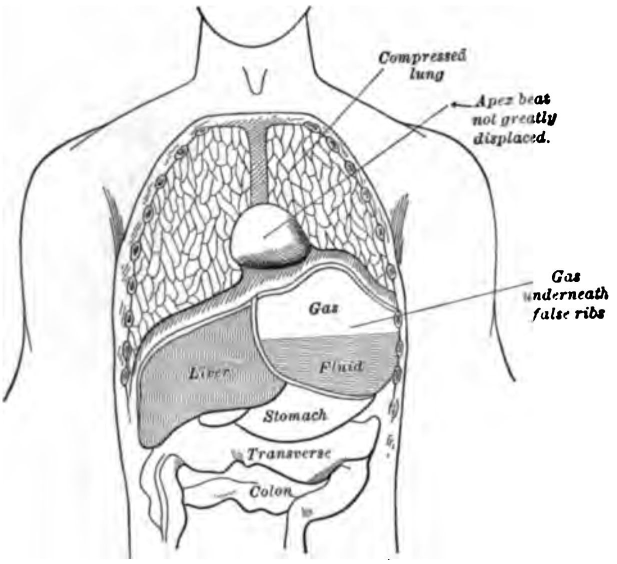


Tumor of the abdomen caused by a dilated stomach. (OSLER.)

Very rarely swelling in the epigastric region, either rapid or slow in onset, follows upon the formation of cysts in the pancreas, as a result of obstruction of a duct of the gland. When they occur, these cysts may quite fill the abdominal cavity, although, as a rule, they are small. As pointed out, however, by Jordan, the cause of a swelling in the pancreatic region may be hemorrhage into the lesser peritoneal cavity. He summarizes some of his views in regard to this matter as follows:

"Contusions of the upper part of the abdomen may be followed by the development of a tumor in the epigastric, umbilical, and left hypochondriac regions. Such tumors may be due to fluid accumulating in the lesser peritoneal cavity, and when the contents are found (on aspiration) to have the power of converting starch into sugar we may assume that the pancreas has been injured." Finally, Jordan states that "many such tumors have been regarded as true retention-cysts of the pancreas."

FIG. 143.



Left pyopneumothorax subphrenicus. (After MAYDL.)

In other instances a swelling in this neighborhood may be due to what is called *pyo-pneumothorax subphrenicus*, a condition of abscess in the peritoneal cavity below the diaphragm, produced by perforation of the stomach or transverse colon or appendicitis. The abscess so produced may contain gas, and for this reason the swelling may be quite resonant on percussion. (Fig. 143.) Abscess in this region also follows abscess of the pancreas or fat-necrosis of this organ in rare instances.

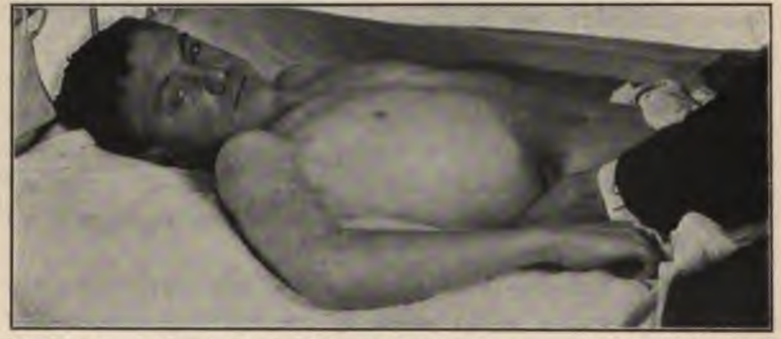
Either palpation or inspection may reveal pulsation in the epigastric area. This may be due to distention or enlargement of the right ventricle or to excessive aortic pulsation or to venous pulsation in the liver. If due to the action of the ventricle there will be additional signs of cardiac disturbance on examining the heart, and in hepatic pulsation there will not only be found tricuspid regurgitation, but a pulsation below the floating ribs at the lower border of the liver. An excessive aortic pulsation is often met with in hysterical or neurasthenic persons without any abdominal lesion. Epigastric pulsation is also often transmitted from the aorta to the hand by enlarged abdominal glands or tumor-masses. If the pulsation of the aorta is not transmitted by glands or tumors, impulse may be due to aneurism of the abdominal aorta, the diagnosis of which is established if, in addition to a pulsating sensation, we also find on palpation a marked thrill, an expansile movement of the tumor, and on auscultation we hear a bruit. Pain due to pressure of the aneurismal sac upon some of the nerves of the abdominal cavity may also be a prominent symptom. Sometimes a horseshoe kidney extending across the vertebral column will mislead one into a diagnosis of an intra-abdominal tumor, for horseshoe kidney is not very rare, being found as often as once in 1650 autopsies.

Localized swellings due to other causes than those already discussed are due to impaction of feces, volvulus and intestinal obstruction from other causes, as, for example, cancer of the bowel. Such a growth occurs most frequently in the cæcum, when the tumor will be found in the right groin, or in the sigmoid flexure, when it will be found in the left groin.

Tumors or foreign bodies in the bowel can nearly always be moved about unless bound down by inflammatory adhesions, so differing from growths which involve the immovable parts, such as the retroperitoneal glands. Very rarely we find a cancerous tumor of the omentum, but when it is present it usually becomes retracted and indurated so that its hardened edges can be felt extending across the abdominal cavity. More commonly when multiple nodules are found in the omentum or studded over the surface of the bowels, they are due to peritoneal tuberculosis. Not rarely these nodular masses are also found studded over the mesentery.

Floating kidney may also cause a marked movable swelling or tumor-like mass in the upper zone of the abdomen. It may be mistaken for a tumor of the uterus, or of the liver, omentum, ovary, or spleen, or even for a much-distended gall-bladder. If the belly-walls are thin, the peculiar shape of the kidney can sometimes be outlined by palpation, and even the pulsation of the renal artery can be felt; but, as a rule, this cannot be done, and the dilatation of the pelvis of the kidney by the obstruction of the ureter, which has become twisted, may distort the shape of the organ. Deep palpation of the flank, if the kidney has floated away from its normal seat, may reveal lessened resistance in this area, and bimanual palpation, one hand being placed at the back and the other in front, may reveal the presence of the organ elsewhere. Further, if the patient be made to lie on the side, the dislocated kidney may sometimes be clearly outlined by bimanual palpation. In other instances, the patient lying on the back with the thighs flexed, the physician grasps the side between the rib and the iliac spine and directs the patient to take a full breath, when the kidney, if movable from its normal resting place, can be felt passing down between the thumb in front and the fingers behind. The fingers should be in the flank and the thumb over the side of the abdomen. (Fig. 144.) If the kidney be pressed upon gently, it can be slipped back into place on expiration. In other instances the patient stands erect, and the physician places the finger-tips of the left hand in the lumbar region, and pressure is made toward the front of the body. The fingers of the right hand are placed anteriorly, and pressure is made backward and upward so as to engage the kidney between the finger-

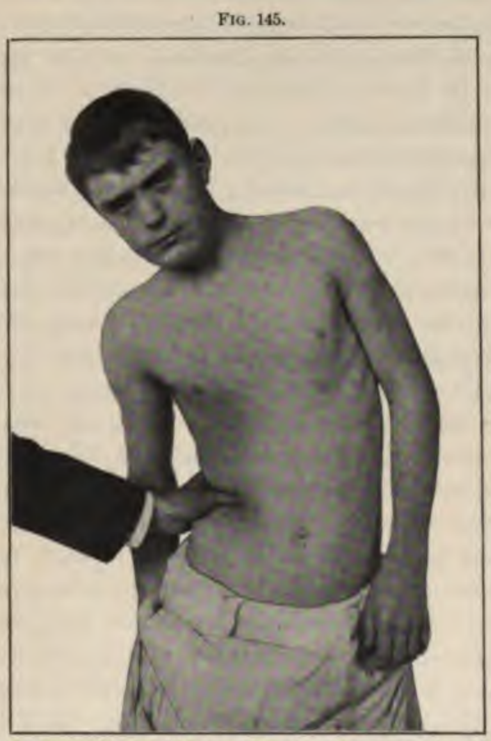
FIG. 144.



Method of examining for floating kidney. The physician grasps the side with his hand in such a way that the fingers and thumb are approximatd, and when the patient takes a full breath the kidney can be felt slipping through between the fingers and thumb.

tips of the two hands. The vertical posture of the body aids in displacing the kidney, which may stay in place in the dorsal decubitus. The kidney may slip with a jerk, if already displaced, back into its normal position; or, if already in place, it may be felt to escape downward into the abdominal cavity. Pressure on a floating kidney causes a peculiar nauseating pain somewhat resembling that produced by squeezing a testicle, and when the organ is caught between the hands it slips from the pressure with a sensation resembling that felt by the fingers when an orange seed is pressed between the finger and thumb and escapes the pressure. The condition of floating kidney is more common in women than in men, but it occurs in both sexes. In 667 cases, 583 occurred in females and 84 in males (Kuttner). It is generally the right kidney which is displaced, although dislocation of the left kidney is not very rare.

FIG. 145.



Method of examining for floating kidney. The physician grasps the side with his hand in such a way that the fingers and thumb are approximated, and when the patient takes a full breath the kidney can be felt slipping through between the fingers and thumb.

In Kuttner's 727 cases it occurred on the right side in 553, on the left in 81, and on both sides in 93. Sometimes violent attacks of renal pain occur in cases of floating kidney. These have been called Dietl's crises. (See chapter on Pain.)

When the kidney is enlarged from cystic degeneration, from ordinary hydronephrosis, and from echinococcus cysts, it may be readily felt in the umbilical area in many instances. Hydronephrosis has been mistaken, in children particularly, for sarcoma of the kidney, and in adult females for ovarian tumor. The diagnosis in some of these cases can be made only by tapping. The fluid obtained in hydronephrosis will usually be somewhat turbid and contain epithelial cells. It should not be forgotten that the condition of hydronephrosis may be intermittent, for, if this is not remembered, the physician may be misled into thinking that the disappearance of the swelling is due to a floating kidney slipping back into its place. This intermittence in the size of the tumor may be of considerable diagnostic aid, for sudden decrease in size would indicate the escape of fluid through a temporarily patulous ureter, and its redevelopment would indicate that this pathway of escape was again closed. Should the fluid escape into the bladder free urination would naturally take place shortly after the tumor decreased in size. Hydronephrosis may be bilateral. In 13 out of 20 cases collected by Roberts this was the case. Severe pain is often a symptom of intermittent hydronephrosis.

A fluctuating swelling in the epigastrium or flank may also arise from cysts of the mesentery. These may grow to a very large size. In other cases a cystic hydroma of the tissues near the kidney may be present. Hawkins has recorded a case in which a large cyst, with an atrophic third kidney attached to it, filled nearly the entire right side of the belly, and from which after death five pints of clear fluid, devoid of albumin and casts, were removed. As already indicated, much diagnostic aid can often be given by tapping an obscure abdominal cyst.

Bulging of the flank, with pain, fever, and perhaps fluctuation, indicates perinephritic abscess or caries of the spine with cold abscess.

In connection with the subject of abdominal tumors, we should not forget the possibility of a floating spleen, a rare condition, but one more common than is generally thought. The shape of the organ, if it can be palpated, will aid the diagnosis, and the presence of resonance on percussion over the area of normal splenic dulness will confirm the diagnosis that the spleen has become displaced. As the spleen in this condition may fall as low as the virgin uterus, it may simulate any growth from a uterine myoma to a tumor of the bowel or pancreas. By reason of twisting of its pedicle and secondary engorgement, its size may be enormous; but if this condition continues, atrophy finally takes place. As such a dislocated spleen drags on the stomach and pancreas, it may cause a long train of curious symptoms, and even intestinal obstruction. Sutton asserts that by pressure it may cause displacements of the uterus.

In this connection J. G. Clark has recommended a method of separating solid from fluid tumors, which is called trimanual percussion. This consists in fixing the mass between two hands, one below it if possible, the other above it. An assistant now percusses, striking the finger of the physician. By this means a thrill can be felt which would otherwise be lost.

"Phantom tumor" is generally found in hysterical women, and often leads to ludicrous errors in diagnossis. It is due to persistent dilatation of a knuckle of intestine by gas, thereby forming a moderately hard and more or less constant mass, which may resemble a real tumor. Examination of the patient under ether will usually reveal its true character. This state is to be differentiated from what Nothnagel has called "intestinal rigidity." In this condition a knob projects through a thin anterior abdominal wall and gradually grows larger until in the course of a few moments it sinks out of sight and touch. It is a disorder of peristalsis. Localized superficial and inconstant tumors may arise through spasmodic but localized contractions of the recti muscles.

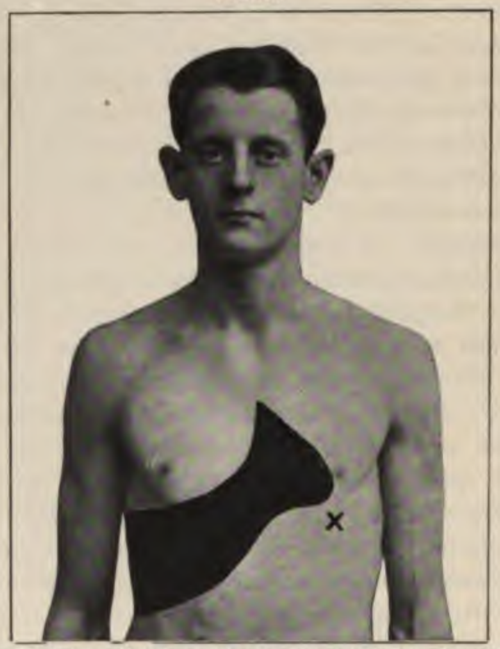
Finally, a swelling in the neighborhood of the umbilicus should always arouse the suspicion of an umbilical hernia. The situation of the swelling at the umbilicus, the fact that percussion over it gives a highly tympanitic note, owing to the gas in the prolapsed gut, and the possibility of reducing its size by taxis in some cases, will render a diagnosis of umbilical hernia possible.

THE LIVER. If on palpating the epigastrium and umbilical area nothing abnormal has been found, we next seek to discover if there is anything abnormal in the right hypochondrium, or, in other words, whether there is any disease of the liver.

Normally, in the adult, this gland cannot be felt below the ribs, except part of the left lobe in the epigastrium occasionally. Sometimes, on deep inspiration, the diaphragm pushes the liver low enough to be felt. In children the liver is naturally large enough to be felt below the ribs.

When the normal liver is percussed we find that it lies in the area shown in Fig. 146, and that as we percuss above it on the ribs in the mammary line we first get pulmonary resonance; then a little below this, impaired resonance, due to the fact that the lower edge of the lung is interposed between the chest-wall and the liver; and still lower we find absolute dulness or flatness, due to the solid liver itself. Below this area, which ceases just below the lowest rib, we usually find tympany on percussion, due to the gas-distended bowel. If we percuss in the midsternal line, we get the same signs; but they begin as high as the nipple, or above it, and then cease at a line drawn across the abdomen about midway between the ensiform cartilage and umbilicus. To the left of the middle line of the sternum the liver-dulness merges into the cardiac dulness. (See Fig.

FIG. 146.

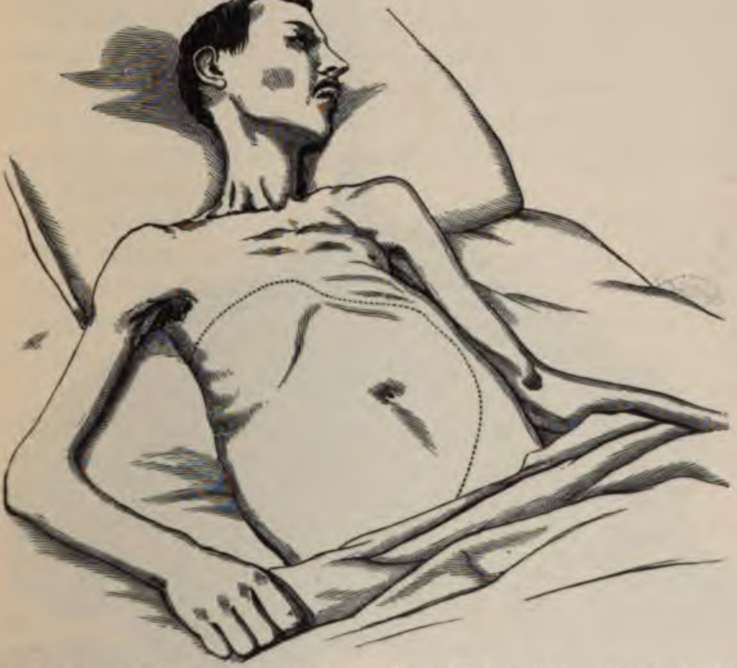


Showing percussion dulness of liver and heart. The outside line shows the area of partial dulness of heart and liver, modified by lung. The solid area is that of true hepatic dulness. A *x*, Traube's semilunar space.

146.) In the mammary line liver-dulness begins at the fifth rib, laterally it begins at the seventh and eighth, posteriorly at the tenth rib, owing to the sloping of the diaphragm.

When a hard and firm mass with a smooth surface can be felt in the right hypochondrium or right umbilical area, which is movable, and which has an edge which can be readily felt on deep palpation, particularly when the patient takes a long-drawn, deep breath, the mass is probably an enlarged liver or a liver pushed down into the abdominal cavity by a large pleural effusion, pneumothorax, a subphrenic abscess, or sometimes by an emphysematous lung. The causes of enlargement are lymphadenoma and amyloid degeneration, congestion, hypertrophic cirrhosis, abscess, carcinoma, sarcoma. When the surface is found to be smooth, the condition is probably amyloid or fatty degeneration, or congestion. If the surface is rough, it will probably be due to cirrhosis, which gives a granular sensation to the hand when the abdominal wall is moved over the organ. In malignant growth large and

FIG. 147.

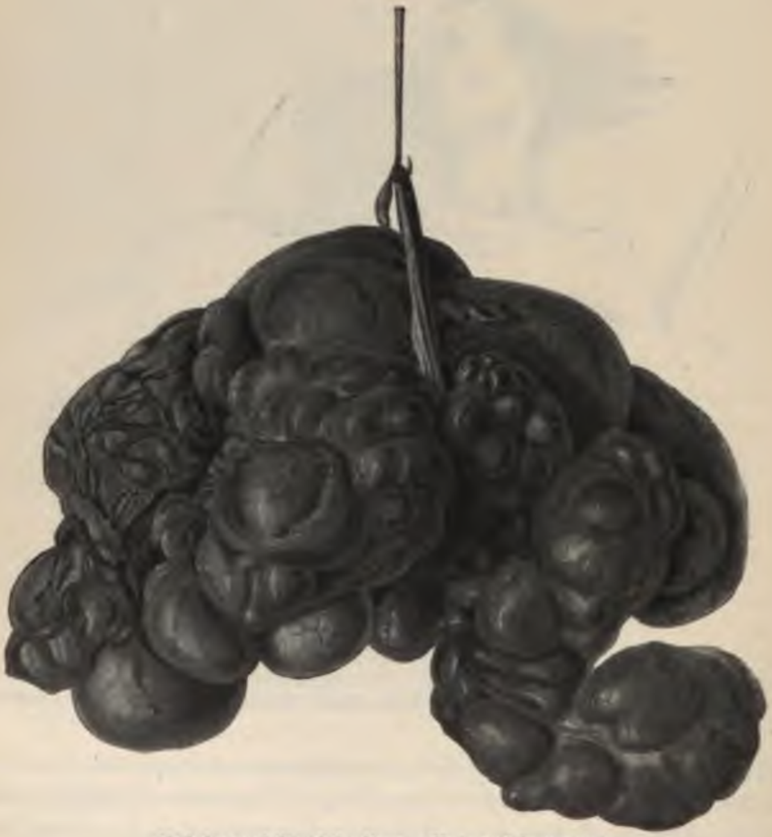


Dotted line shows area of cancerous liver extending far beyond its normal area. Over the entire surface of this mass could be felt hard nodular masses. (From the author's wards, Jefferson Medical College Hospital.)

small nodules may often be found, and depressions or umbilications of its surface may be marked, but it must be remembered that cancer of the liver is not necessarily associated with the presence of nodular masses. On the contrary, the growth or growths may be large, yet project so slightly above the hepatic surface that they cannot be felt. In such cases there may be pain, marked emaciation, cachexia, and the organ be found much enlarged. (Fig. 147.)

The physician who feels distinct nodules on the surface of the liver should not immediately conclude that these are necessarily carcinomatous, for syphilis often produces a very extraordinary nodulation of the surface of this organ. So great is this, that when nodulation is excessive the possibility of syphilis being the cause is to be

FIG. 148.



Nodular syphilis of the liver. (KAST and RUMPLER.)

considered. This form of disease is, however, rarely accompanied by as great hepatic enlargement as is that due to cancer with marked and multiple nodules. (Fig. 148.)

The consistency of the liver is usually very hard in cases of cirrhosis, carcinoma, and amyloid degeneration. In cirrhosis there will be some ascites in many cases, some swelling of the legs perhaps, and dull pain in the hepatic region. The digestion will be disordered, there will be marked loss of flesh, and often hæmatemesis. Sometimes coma comes on. In the case of amyloid liver there will be a history of prolonged suppuration elsewhere, and there will be present disordered digestion, irregular bowel-movements, and little pain.

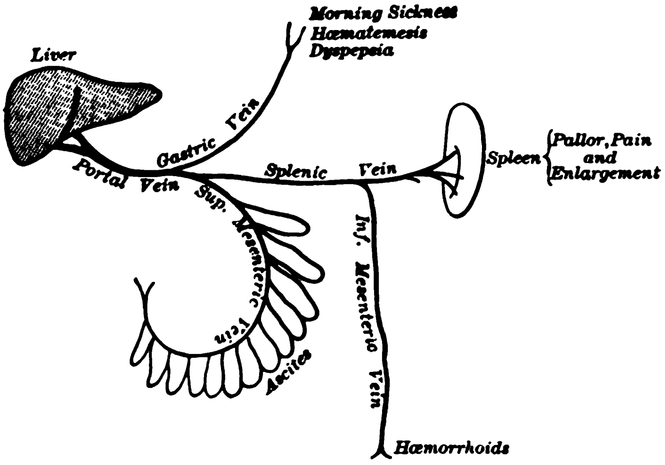
Marked tenderness of the hypochondrium is usually found in congestion of the liver, in inflammation of its tissues, such as that caused by an infection or by gallstones in its substance, and in malignant growth. Tenderness is practically absent in amyloid liver and in fatty degeneration.

In cases of cirrhosis of the liver, whether it be in the hypertrophic or atrophic form, the patient rarely complains of the organ, and no symptoms which seem to him hepatic in origin may be present, save that in the hypertrophic state its size is increased, so that it can be felt below the ribs, whereas in the atrophic state it cannot be felt except by pushing the fingers well up under the ribs. The symptoms accompanying cirrhosis are chiefly connected with disorders of the alimentary canal, either through direct failure in the digestion and assimilation of food, or from changes in the blood-supply of the abdominal contents. The following excellent diagram, from Seymour Taylor's *Index of Medicine*, shows what these symptoms are, and discovers their cause at a glance, the cirrhotic process, of course, obstructing the flow of blood in the liver. (Fig. 149.) It is a noteworthy fact that in the atrophic form jaundice is rare even in the very last stages of the disease, whereas in the hypertrophic form it is commonly met with. Ascites is common in the atrophic form, rare in the hypertrophic variety.

Sometimes enlargement of the liver and ascites are due to adhesive pericarditis. The diagnostic signs of adhesive pericarditis consist in systolic retraction of the intercostal spaces in the anterior axillary line, and posteriorly at about the fifth or sixth rib on the left side, which retraction is followed by a diastolic rebound. If the patient is told to forcibly inspire or expire, the natural change in the position of the apex-beat of the heart in relation to the chest-wall does not occur, neither does the edge of the lung on full inspiration diminish the area of cardiac dulness as it does in health.

The physician who finds the lower margin of the liver abnormally low down in the abdominal cavity should not make a diagnosis of enlargement of this organ until he has assured himself that the extension of the margin of the liver is not due to an effusion in the right pleural cavity which presses upon this organ. So, too, if the patient is a woman, the lower border of the liver may have been pushed down by tight lacing, and careful palpation may reveal a furrow across its surface produced by the corset. The displaced lower border of the gland gives rise to the diagnosis of enlargement of the liver if careful percussion shows that the upper border of liver dulness is in its normal place. If the upper border of the liver is depressed, and pleural effusion is absent, it is a sign of floating liver.

FIG. 149.



To illustrate symptoms of cirrhosis of liver. (SEYMOUR TAYLOR.)

When, on palpating the liver, we find marked tenderness and some swelling, and, associated with these symptoms, fever, rigors, sweats, and sometimes vomiting, and, in addition, a history that the patient has had dysentery or has had exposure to tropical heat or has swallowed bad water, the presence of an abscess of the liver is indicated. This may be single or multiple. If the latter, it is probably due to pyæmia, and no spot of fluctuation will be found, as a rule; whereas, if it is large and single, fluctuation is sometimes felt. Further, the enlargement of the liver in the pyæmic form is uniform, whereas in the single abscess there is often one spot which is swollen or enlarged. The history of the case will usually separate the conditions, one from the other, for diagnostic purposes, for in the case of abscess the history will probably be that of a person exposed to tropical heat or one who has had an injury, an acute infection, or an amœbic dysentery. More rarely a single hepatic swelling may be due to hydatid cyst, but the history and presence of fluctuation, combined with the result of examining the fluid aspirated from the swelling, will decide the diagnosis. Further than this, hydatid cyst yields on percussion a peculiar vibratory thrill called the hydatid thrill. Three fingers are placed over the area, the middle one being pressed firmly upon the growth and the lateral ones but lightly. The middle finger is now percussed with the other hand and allowed to remain *in situ*, when an after-thrill may be felt in the other fingers.

While palpating the lower border of the liver, the physician may feel a mass projecting below its margin. If the mass is of good size and the abdominal wall thin, it will be felt as a pyriform body and may be slightly fluctuating. Such a mass is probably a distended gall-bladder, and it may be associated with enlargement of the liver and with jaundice. The causes of enlargement of the gall-bladder are several, viz., distention from the accumulation of bile produced by obstruction in the cystic or common duct, or the presence of a large number of calculi in the gall-bladder, and often to the accumulation of thin mucus, which may or may not be bile stained, the so-called hydrops of the gall-bladder. The causes of acute obstruction of the ducts are gallstones, new growths, such as carcinoma and rarely gumma, and inflammatory processes in the ducts themselves, with inflammation in surrounding tissues and organs. Often the inflammatory processes just named will have been produced by the presence of gallstones, which may, by causing ulceration of the mucous membrane, permit severe infections to take place. Rarely is it due to ascarides or echinococcus (Courvoisier states the proportion to be in 74 cases, 57 due to stone, 7 to ascarides, and 3 to echinococcus). The question then arises as to the cause of the enlargement in the particular case at hand. Obstruction of the bile-ducts is, as already related, due, in the vast majority of cases, to stone, aside, of course, from that due to an acute catarrh which is but temporary in its effects, and therefore the probable cause in every case is stone. If this is the cause, the patient will probably give the history that at some time in the near or remote past, after a period of more or less distress in the hypochondrium, she has been seized by a pain which was paroxysmal in type and extended backward and upward to the region of the scapula or shoulder. This pain was also peculiar in that it was apt to occur at night rather than after taking food, as in gastric cancer or ulcer, because the bile is flowing to the gall-bladder at this time, instead of directly into the bowel. Examination at such times may have revealed a swelling of the gall-bladder. There may or may not be a history of jaundice. Manifestly this is a history of an attack or attacks of gallstone colic, and should lead us to the belief that the enlargement of the gall-bladder is due to stone. In this connection it is well to take into consideration the importance of the presence or absence of jaundice at this time, and this is well expressed by Mayo Robson in the words, "jaundice with distended gall-bladder is presumptive evidence of malignant disease, but jaundice without distended gall-bladder favors the diagnosis of cholelithiasis. This is sometimes called "Courvoisier's law." In other instances there may be no history of attacks of gallstone colic, the patient simply complaining of pain and discomfort in the hepatic area, and it is important to remember that in some cases of enlargement of the gall-bladder due to obstruction of its duct the enlargement may be so gradual and so great that the distended viscus may be felt far from its normal site in the middle line or even under the left ribs.

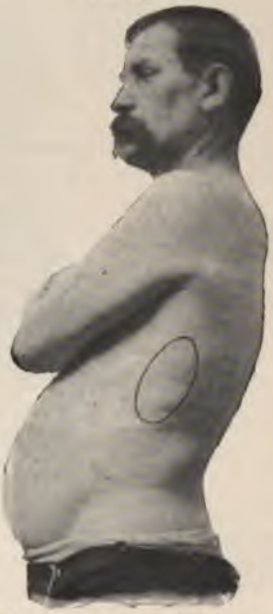
An additional symptom favoring stone as a cause is fever, which is due to infection produced by the stone injuring the mucous membrane, and so producing septic absorption, the micro-organisms passing up from the bowel. Sometimes it is simply an indication of inflammation, at others it indicates anything from mild purulent infection of the bile-passages to empyema of the gall-bladder or a general cholangitis. If the infection be with a benign organism, the symptoms may be mild; but if it be due to the streptococcus, it is often virulent, and the febrile movement most severe. Much depends, too, upon the freedom of drainage. If the gall-ducts permit the escape of the pus into the gut, the symptoms may become greatly reduced; but if it be retained they are apt to become more pressing. (See Cholangitis, in chapter on Fever.)

A new growth by pressing on the gall-ducts may obstruct the flow of bile and produce jaundice, and a mass; but there is usually no paroxysmal pain, and the growth is usually secondary to a growth elsewhere in neighboring parts, which may be demonstrable. Rare as is primary malignant new growth of the gall-bladder, it is almost unknown in the liver, but it may be secondary to gastric carcinoma.

The general cachexia, rapid loss of weight, and the age of the patient may aid the diagnosis of the tumor, and in this respect Courvoisier's law is to be recalled. (See p. 354.)

When the gall-bladder is distended with gallstones there is usually a history of colic; there may be gallstone-crepitus on careful palpation, and symptoms of fever due to a cholecystitis set up by the stones. However, it is not to be forgotten that gallstones are present in about 10 per cent. of all adults, although, as Kehr has well pointed out, fully 95 per cent. of such persons come to autopsy from other causes without even suffering from any manifestation of their presence. It is only when the calculus produces irritation or mechanically blocks the ducts that symptoms develop. As already stated primary cancer of the gall-bladder is usually due to the irritation produced by gallstones. (See chapter on Skin, Jaundice.) Tenderness on pressure will be marked. Sometimes ascites will be present, and a growth may be found, usually as the primary lesion, in the stomach or bowel.

FIG. 150.

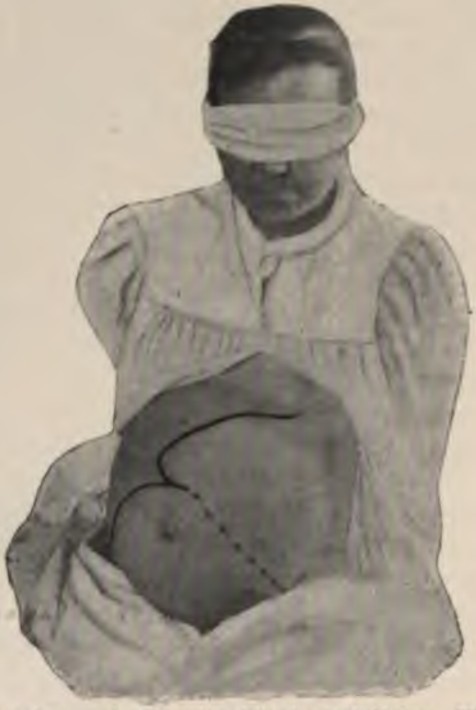


Normal position of the spleen.

THE SPLEEN. In the left hypochondrium the spleen can be very readily outlined by percussion in persons not inordinately fat. Its normal position is best shown in the accompanying figure. (Fig. 150.)

The upper border of the spleen is on a level with the tenth dorsal vertebra and the lower border on a level with the end of the eleventh rib. Its upper edge or limit is on a level with the ninth rib. In percussing the spleen heavy percussion is to be avoided, since this may develop the resonance of the stomach or bowels. The spleen cannot be palpated unless greatly enlarged, but it may be found bulging from beneath the lowest rib in typhoid fever (Fig. 151); in scarlet fever; as the result of acute or chronic malarial fever; in leucocythæmia of the spleno-medullary variety (Fig. 152) (see chapter on Blood); in amyloid disease, as that after long suppuration; in early syphilitic infection; and in any disease which causes venous engorgement of the abdominal viscera, such as cardiac disease or hepatic cirrhosis. The spleen sometimes reaches a very large size in the disease which has been called "splenic anæmia." In this condition we have a state resembling Hodgkin's disease, save that there is not the involvement of the lymphatic glands. The blood, unlike that in true leukæmia, does not show great changes in the white cells; any changes present, consisting in an increase in the relative number of the lymphocytes which may, in some cases, make up nearly all of the leucocytes. The red cells are greatly decreased in number, and

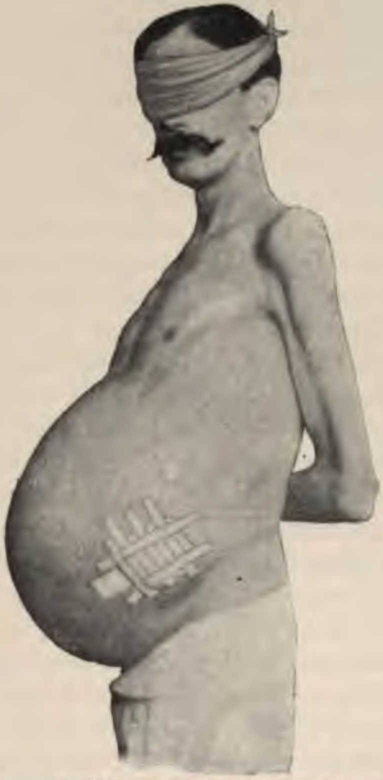
FIG. 151.



A case of chronic enlargement of the spleen following typhoid fever. The dark line shows the margin of the organ on palpation, while the retraction in the line and the dotted line indicate the position of the splenic notch. (From the author's wards in the Jefferson Medical College Hospital.)

there are present poikilocytes, megalocytes, and microcytes. Closely allied to this condition of the spleen is that which consists in the so-called "Banti's Disease," in which the spleen is also greatly enlarged. This disease is divided by Banti into three stages: a stage of anæmia characterized by enlargement of the spleen, and lasting from three to ten years; a transitional stage; and a third stage of marked ascites, which usually terminates in a few months. In some cases of so-called Banti's disease, hemorrhages take place, such as vomiting of blood; nose-bleed, hæmoptysis, and hæmaturia. Banti's disease differs from spleno-medullary leukæmia in the absence of an excess of white cells, and it is supposed to differ from splenic anæmia in that there is not the same increase of lymphocytes as is characteristic of that condition. It is to be remembered that splenic anæmia and Banti's disease are scarcely regarded as pathological entities by

FIG. 152.



A case of enormous enlargement of the spleen in splenic leukæmia. (From a private patient.)

many clinicians. These terms cover obscure conditions about which we know very little.

Sometimes displacement of the spleen downward arises from emphysema of the lungs or left-sided pleural effusion.

Acute splenic swelling sometimes comes on in cases of general septicæmia.

Nearly always the splenic surface is smooth, except for the notch in its surface (see Fig. 151), unless the disease be the rare condition of hydatid disease or carcinoma.

In cases in which it is difficult to determine the location of an abdominal growth it may sometimes be localized by distending the colon with air, which is gently injected into the bowel by an ordinary bulb syringe.

There yet remains for discussion the significance of increased resistance on palpation, and percussion-dullness, in the groins. In the right iliac region the presence of swelling, increased resistance, impaired resonance, or tympanites, particularly if pain and tenderness are present, point strongly to appendicitis or to inflammation about the *caput coli*. Sometimes, however, the presence of a distinct lump in this region in a person advanced in life may mean a malignant growth, for carcinoma of the caput coli is not rare.

If the left groin is affected in a person well advanced in years, carcinoma is also to be regarded as possible, for the sigmoid flexure is a frequent seat of such growths. In a young person or a child impaction of feces, a foreign body, or intestinal obstruction is also to be considered. (See chapters on Vomiting and on the Bowels.)

For the diagnosis of renal disease reference is to be made to the chapters on the Bladder and Urine, the Blood, the Bloodvessels and and the Pulse, and upon the Thorax (that part on the Heart), to the chapters on Vomiting and on Headache, and to those on Coma and Unconsciousness, and Convulsions and Spasms.

For further information in regard to the diagnosis of disease of the abdominal viscera, the reader is referred to the chapter on the Skin (that part on Jaundice), the chapter on Vomiting (part on Intestinal Obstruction), to that on the Bladder and Urine, and to that on the Bowels and Feces.

CHAPTER IX.

THE BLOODVESSELS AND PULSE.

The condition of the bloodvessels on palpation—Feeling and counting the pulse—The quality, force, and volume of the pulse in health and disease.

One of the first things that the physician does when he is studying the condition of a patient is to feel the pulse, even if the symptoms which are present do not indicate circulatory disturbance, because the pulse is an index of the condition of the heart as to its power, its valvular action, and its nervous state. The pulse very often gives us information of the presence of renal disease, and it will frequently give us a general idea of the tone or degree of debility of the patient. By feeling the pulse we also gather valuable information as to the condition of the arteries, and this is a very important part of the diagnosis, for, to use an old saying, "a man is only as old as his arteries;" and if he is sixty years of age and has good vessels, he is, as a rule, as young in health as another man of thirty with bad vessels, because it is by the bloodvessels that the tissues of the body are nourished, and, as life depends upon this process of nutrition, the better the vessels the better the vitality.

When examining the pulse of a patient who is well enough to be up and about, the physician should wait until sufficient time has elapsed after exercise for the pulse to become steady, and the patient should be in a sitting or reclining posture in order to prevent overaction of the heart. Particularly is it important in the case of nervous individuals to wait for sedation to follow the excitement of meeting the physician.

Often when called to see a sick child or a nervous woman, who may be sleeping at the moment of the physician's arrival, a true estimate of the pulse can be made without disturbing the patient by gently putting the tip of the finger on the temporal artery where it passes over the zygomatic process. This fact is of considerable importance, because in some patients the excitement of the doctor's visit may produce marked alterations in pulse-rate. This artery may also be used for this purpose in cases of tremor, chorea, delirium, or mania, in which the hand is constantly moved about so that the radial artery cannot be felt.

In counting the pulse it is best to count it for the entire minute, or to count it for fifteen seconds and then multiply the result by four. If the pulse is irregular, it is always best to count it for a minute. If the pulse be very irregular and running, and so difficult of counting, the count should be made by listening at the præcordium for the apex-beat.

Before considering the qualities of the pulse in health or disease, it is well to understand what it is due to and the manner in which the circulation is carried on. The bloodvessels consist of the arteries, arterioles, capillaries, venules, and veins. These vessels also contain blood during life, and the function of the heart is to propel the blood through them. The flow of blood is maintained, first, by the force expended by the heart, and, second, by the tonicity of the bloodvessels. If the bloodvessels of the body become relaxed, as in death, all the blood is readily held by the ones most relaxed, namely, the abdominal, thoracic, and other veins. We find, therefore, that the vessels are only filled with blood when their walls are to a certain extent constricted by the contraction of their muscular fibres; and that this contraction is maintained by the action of the vasomotor centre in the medulla oblongata, which also controls many minor centres governing small areas of vessels. The arteries are very elastic in health, and when filled with blood are slightly distended. Behind the column of blood, which being a fluid confined laterally is practically a solid, for fluids are incompressible, is the heart, and in the arterioles are muscular fibres, which by their contraction regulate the flow of blood into the capillaries, from which the nutritional processes are carried on. The blood in the arteries is, therefore, subject to three chief pressures, namely, that of the heart behind the column, that of the elastic and distended arterial walls on the sides of the column, and the resistance of the contracted arterioles in front of the column. By these means blood-pressure or tension is maintained. If the heart beats more strongly or the arterioles contract more tightly than normal, the blood-stream is under a greater pressure than before. If the heart is feeble or the arterioles lax, the pressure falls, because the blood is not pressed upon behind or obstructed in its flow in front. If the tension is above or below normal, the interchange of food and oxygen and carbonic acid between the tissues and the blood in the capillaries is perverted, for the rate of flow in the capillaries depends largely upon the blood-pressure in the arteries. As the capacity of the capillary system of vessels is many times greater than that of the arteries, if the arterioles relax, the capillaries and veins will retain all the blood, and it will stagnate and become useless.

The manner in which arterial tension is chiefly maintained having been described, we can now consider the pulse-beat itself. The individual pulse-beat is not a wave of blood sent out by the heart, but it is the transmission of the force of the heart-beat sent along the blood-column, and the character of the beat gives us, therefore, an idea of how forcibly the heart is driving another quantity of blood into the aorta, and also how much blood is being sent out at each beat.

Supposing, therefore, that on feeling the radial pulse we find that the artery is tense and hard, and that the individual beat is strong and its volume great: this signifies that there is an excited vasomotor centre, causing contraction of the vessels, and that an excited, over-acting heart is forcing the blood into the already tense vessels.

If this condition increases, one of three things can happen: either the heart will be unable to pump the blood out into the arteries against the pressure, and consequently become distended and enfeebled, or a bloodvessel will burst in its weakest spot, or the spasm of the arterioles will give way. It is the first result which we meet in cases of true angina pectoris, for in this state we find great arterial tension, with distention and engorgement of the left side of the heart, and the moment nitroglycerin or nitrite of amyl relaxes the spasm of the arterioles the symptoms are relieved. It is the second result which often produces apoplexy by rupture of the weakest vessel, usually the middle cerebral artery. It is the third result which we try to bring about for the relief of the patient, either by drugs or by bleeding. When we have atheroma or hardening of the bloodvessels as the result of old age, syphilis, or other chronic vessel-changes, the very inelasticity of the vessels, associated, perhaps in some cases, with some irritability of the vasomotor centre, causes a high arterial tension, with a laboring heart, a congested head, and a feeling of fulness of the head, of which the patient will complain. The second sound of the heart will also be much accentuated.

The discovery of a high arterial tension in a young person, or in an older one who has not atheromatous vessels, will generally mean the presence of an excited circulation, in connection with some acute inflammatory disease in its early stages, and, if high fever is present in a previously healthy person, the pulse will be found to be quick and hard.

One of the most important acts of the physician is to make a skilful determination of the arterial pressure of his patient, since this is of even greater importance than the study of the pulse-rate. Years of experience enable the physician to determine the tension of the arteries by his finger-tips, and often the degree of tension gives him far more information than any other physical sign presented by the patient. A persistent high arterial tension, in the absence of fever, usually indicates chronic-contracted kidney, although a high tension is found in some cases of parenchymatous nephritis, and in instances where atheromatous changes have taken place in the heart and bloodvessels from other causes. In acute fevers in the early stages the tension is often unduly high. Efforts have been made by numerous investigators to devise apparatus which would be capable of indicating the condition of arterial tension, but all of them have been more or less unsatisfactory. Probably the best at present devised is the tonometer of Gaertner. This consists of a pneumatic ring of rubber placed inside a metal ring which is connected with heavy rubber tubing and air-tight joints with a T tube, one arm of which is attached to a mercury manometer, and the other arm to a hollow rubber ball without a valve. The ring is slipped on to one of the fingers as far as the second phalanx, the end of the finger is then rendered bloodless by a special compressor or rubber band, and the pressure of the whole apparatus is raised to a point considerably above the blood-pressure by squeezing a rubber ball. After this the compression band is removed and the pressure gradually reduced until at a particular instance the patient feels a throb and the examiner sees the pallid finger-end suddenly redden. The reading of the manometer is then taken, and represents the pressure just below that needed for obliteration of the digital arteries. The advantage of Gaertner's apparatus over the others which have been invented are its simplicity, its moderate cost, and its approximate accuracy.

A condition of intense vascular relaxation, due to failure of the heart or the arterioles to maintain blood-pressure, is seen in cases of fainting and syncope on the one hand, or of collapse and shock on the other. Here we find a soft, easily extinguished blood-stream, which can, by pressure on the artery, be readily cut off from the distal vessels. The artery feels relaxed to the physician's finger and the skin may be bedewed with sweat.

A very low arterial tension indicates a feeble condition of the system, such as is seen in all exhausting diseases, acute or chronic, or, if no disease be present, in the sense of an acute malady, it indicates general nervous debility, with or without the presence of a feeble and dilated heart. It is often met with in mitral stenosis and indeed in all forms of cardiac disease with ruptured compensation.

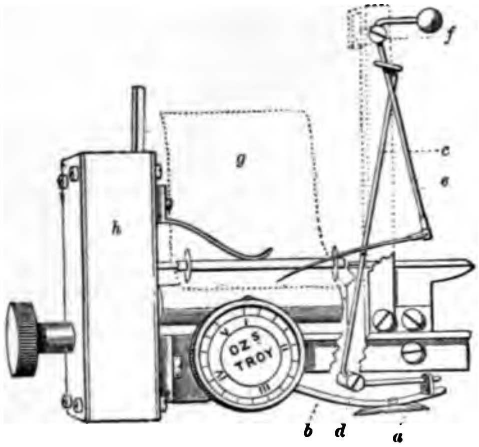
The pulse itself varies as to volume, character, rapidity, and force, and does so within normal limits, and still more so under the effects of disease. It varies greatly according to age. Thus, the pulse of the newborn child is usually about 135 to 140, at one year 120 to 130, at two years 105, at four years 97, at ten years about 90, at fifteen 78, and from twenty to fifty years about 70 per minute. At eighty years of age it is usually about 80 beats per minute. The rate is also increased by taking food, by exercise, by nervousness, and by pain and fever, as will be stated again in a moment.

The volume of the pulse-wave depends chiefly upon the quantity of blood expelled from the heart at each systole, and also upon the condition of the aortic valves of the heart, in so far as their ability to prevent regurgitation is concerned. The stimulation of the vagus nerves usually results in a large pulse-wave, as does also cardiac hypertrophy with dilatation. If, on the other hand, part of the blood thrown out of the heart into the aorta falls back into the ventricle, we have a pulse of small actual volume, and this is called, because of the peculiar sensation which it gives to the finger, "trip-hammer," "water-hammer," or "Corrigan's pulse." In such a case, because of the power of the ventricle, the blood is forced out into the aorta with great force, but as the last part of the wave regurgitates the pulse is found to be short and sharp. In mitral regurgitation or in mitral stenosis the pulse is usually small in volume, because the left ventricle has not, or cannot get, enough blood at each beat to send out a voluminous wave. (See Figs. 155 and 156.)

So far as the character of the pulse is concerned, we recognize one which is slow and full, as that seen after digitalis is used; that which is short and sharp, as in aortic regurgitation; that which is small and hard, as is often seen in aortic obstruction, and the small, wiry pulse of acute peritonitis.

Various names are applied to a pulse possessing certain peculiarities. Thus, we have under the name *pulsus paradoxus* a pulse which disappears with each deep inspiration. It is usually due to indurative mediastino-pericarditis, whereby inflammatory bands press on the bloodvessels or the heart. If the beats of the heart are irregular in force but regular in rhythm, we have developed what is called a *pulsus alterans.*

FIG. 153.



A sphygmograph. (DUDGEON'S.) Certain supporting parts are omitted so that the multiplying levers may be displayed. *a* is a small metal plate which is kept pressed on the artery by the spring *b*. The vertical movements of *a* cause to-and-fro movements of the lever *c* about the fixed point *d*. These are communicated to and magnified by the lever *e*, which moves round the fixed point *f*. The free end of this lever carries a light steel marker which rests on a strip of smoked paper, *g*. The paper is placed beneath two small wheels and rests on a roller which can be rotated by means of clock-work contained in the box *h*. The paper is thus caused to travel at a uniform rate. The screw graduated in ounces (Troy) is brought to bear on the spring *b* by means of a cam, and by this the pressure put on the artery can be regulated. The levers magnify the pulse movements fifty times.

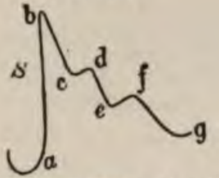
A *dicrotic pulse* is one which is characterized by a reduplication, which feels like a second beat following the first before the latter is over. It is found in many cases of exhausting fever, and depends upon an undue elasticity of the bloodvessels, with relaxation of the arterioles, so that the blood first unduly distends the arteries, which then contract upon it, and thus produce the second wave or apex to the pulse-curve.

We can study the pulse either by the touch or by the sphygmograph. If by the latter means, the instrument of Dudgeon is the best. (Fig. 153.)

The normal pulse-wave is shown in Fig. 154.

It will be seen that there is a distinct upstroke produced, which is called the line of ascent. This is due to the distention of the artery produced by the ventricle forcing blood out into the aorta. There is after this a line of descent interrupted by two separate secondary waves, which are called catacrotic waves. The second or lower of these is called the dirotic wave, and is the one which becomes marked enough to be felt in some cases of disease. The duration of the period of descent corresponds to the time the blood is flowing out of the arteries into the capillaries, and, if this flow is rendered difficult by vascular spasm, the line of descent will be gradual; if easy from vascular relaxation, it will be short. If the drop is very sudden, it is a pulse of "empty arteries," so called, as after severe hemorrhage or in cases of aortic regurgitation.

FIG. 154.

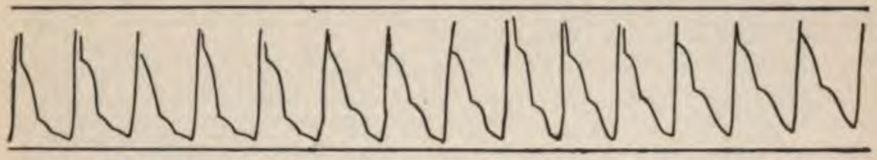


a b. Percussion upstroke. *a b c.* Percussion-wave. *c d e.* Tidal wave. *e f g.* Dicrotic wave. *d e f.* Aortic notch. *f g.* Diastolic period.

Very small irregularities of the line of descent are due to the elastic bloodvessels being thrown into vibrations by a forcible pulse-wave.

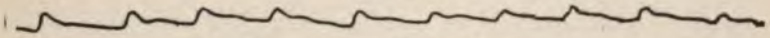
In Fig. 155 is shown the typical pulse-wave of aortic regurgitation; and in Fig. 156 that of mitral stenosis, which is irregular in time and volume. (See page 363.)

FIG. 155.



A tracing taken from a case of aortic regurgitation. Corrigan's pulse. Note the sharp upstroke and the sudden fall due to the blood falling back into the ventricle.

FIG. 156.



The small pulse of mitral stenosis.

The rapidity and force of the pulse also depend largely on the condition of the bloodvessel-walls, particularly the rapidity. The latter also is influenced by the activity of the pneumogastric nerves in regulating the beat of the heart. Thus, if the arterial pressure be very high, through spasm of the arterioles, the difficulty experienced by the heart in forcing blood into the arteries will be so great that pulsation will be very slow, whereas if the normal resistance to the action of the heart be removed by vasomotor relaxation, the beat will be rapid, just as the wheels of a locomotive fly around on a slippery track when the friction or resistance is removed. If the vessels are relaxed, the impetus communicated to the column of blood in the vessels by the heart is lost, and so the pulse is not forcible; or if the resistance is excessive, the force is dissipated.

The vagus or pneumogastric nerves are continually holding the heart in check, and by causing full diastole enable it to send out a large wave of blood at each contraction. If they are greatly stimulated, we have a very slow pulse and a full wave of blood with each heart-beat; but as the heart now beats very slowly the blood-pressure may fall for lack of blood in the vessels, unless there is an increased force of the heart at each contraction to make up for the number of beats in the minute which have been lost, or unless there is also a great increase in arterial tension by contraction of the vessels. A very slow pulse depends in the great majority of cases upon a high arterial tension from vascular spasm—*i. e.*, resistance to the flow of blood; more rarely it is due to irritability of the vagus nerves, produced by pressure or disease, or by drugs, such as digitalis.

The term bradycardia is applied to a very slow pulse, which may be as slow as twelve beats a minute.

A rapid pulse is seen most commonly as the result of stimulation of the heart by drugs, by fever, or by fear. Fear causes the vagus to lose control of the heart, and fever acts by reason of the stimulant effect of heat upon this viscus and its depressant effect upon the vagus. In other words, the quick pulse of fever is not a mere coincident symptom of fever, but the result of it.

When the heart's action becomes exceedingly rapid it is called tachycardia. It is due in the majority of instances to relaxation of the bloodvessels, and more rarely to depression of the pneumogastric nerves. As a symptom of organic disease it is a frequent manifestation of exophthalmic goitre. Often in this condition the pulse becomes so fast that it cannot be counted.

Great force of the pulse is due to hypertrophy, or over-action of the heart because of stimulation; and great feebleness is generally caused by marked dilatation not associated with hypertrophy, by chronic myocardial degeneration, and in acute disease by exhaustion of the heart-muscle, or changes in the muscle-fibres of the heart.

CHAPTER X.

THE BLOOD.

The various forms of red and white corpuscles—Their proportionate number in health and disease—Alterations in their form and character—The hæmoglobin of the blood in health and disease—The various forms of anæmia—Leukæmia and pseudo-leukæmia—Parasites of the blood.

As already pointed out in the chapter devoted to the skin, marked changes in the blood speedily produce manifest alterations in the appearance of the patient. The present chapter will be devoted to a consideration of the changes in the blood seen by the naked eye or by the aid of various forms of delicate apparatus designed to give us accurate observations. Before studying the conditions of the blood which are found in disease, it is well to briefly rehearse the characteristics of normal blood when it is examined outside the body.

The blood consists of a liquid basis or plasma, in which are found two varieties of cells—the red blood-cells and the white blood-cells. The red cells are called erythrocytes, the white cells leucocytes. The red cells are biconcave disks, dark at the edges, and with a clear or bright spot in the centre, due to their biconcavity. They do not contain a nucleus. The red color of the blood is due to the aggregation of immense numbers of these bodies, the coloring-matter of which is called hæmoglobin; but if a few corpuscles be placed in a bright light on the stage of the microscope, they look bright and yellow. The number of red blood-cells is about 5,000,000 to the cubic millimetre of blood in a healthy adult male, and about 4,500,000 in the healthy female. If this number is exceeded, which is very rare, the condition is called polycythæmia; if decreased, the condition is called oligocythæmia. One of the most marked instances of polycythæmia which occurs is the very extraordinary increase of red blood-cells which is often met with in cases of congenital cardiac disease in children, amounting to as many as 8,000,000 to the cubic millimetre. A similar increase is seen in phosphorus-poisoning. Beside the ordinary red blood-cells we find in health small red cells supposed to be immature red cells and [illegible] we find also [illegible] may be the immedi-[illegible] hæmoglobin may [illegible] per cent. of hæmo-globin [illegible] when the hæmoglobin is [illegible] per cent. [illegible] hæmoglobin [illegible] anæmia.

In disease we find [illegible] alterations in the red cells themselves and in their [illegible]. The [illegible] and mega-[illegible] great in number. [illegible] are found in great number and are [illegible]. Thus red cells which, unlike ordinary healthy cells, possess a nucleus and are capable of [illegible] movement, are found and are called [illegible] name of [illegible] and finally we find other red cells [illegible] or again [illegible] in [illegible] to be called [illegible]. The diseases in which these changes are found will be discussed further on.

The proportion of the white to the red cells in health is about 1 to [illegible], [illegible] great variations occur. Thus after meals the white corpuscles are [illegible] increased so that the proportion may be 1 to [illegible]. On the other hand after this primary increase, they may be decreased and the proportion may be 1 to [illegible]. Time of day [illegible] a factor in producing a variation. [illegible] found before breakfast [illegible] one hour after breakfast, 1 to [illegible]; three hours after breakfast, 1 to [illegible]; ten minutes after dinner, 1 to [illegible]; half an hour after dinner, 1 to [illegible]; two and a half hours after [illegible] 1 to [illegible]; half an hour before supper, 1 to 544; and [illegible] after supper, 1 to [illegible]. It is important to remember that [illegible] of food [illegible].

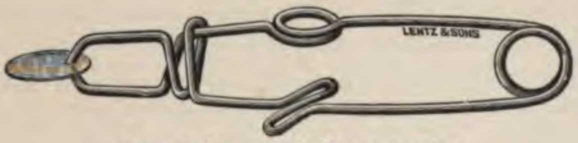
Not only may these causes produce great alterations in the results obtained in a blood examination, but many others must be considered, [illegible] always bear in mind that there are great variations in the blood within healthy limits, both in quantity, quality, and corpuscular parts. For these reasons slight variations should not be given much weight unless they are found constant by repeated tests. It is usually unsafe to rest upon a single test. Thus sweating, if profuse, may cause such a concentration of the blood that a normal number of red cells to a cubic millimeter may be present when in reality there is marked anæmia. So, too, tapping an effusion, or the use of an active hydragogue cathartic, or profuse diarrhœa may greatly increase concentration. Blood obtained from a cold ear or a cold finger-tip, or from these parts when made unduly warm by friction or heat, gives erroneous ideas of the state of the blood in general.

Proceeding, then, to the study of the blood for diagnostic purposes, we examine it by the microscope, and by color-tests. The object of the microscopic examination is to determine the quality and character of the red and the white corpuscles, their number, and the presence of parasites. The color-tests are for the purpose of determining the proportions of hæmoglobin, or, in other words, the ability of the corpuscles to carry oxygen to the tissues.

To study the blood microscopically we need a quarter-inch objective for ordinary corpuscular work, or, as they say on the Continent of Europe, a Number 7 Hartnack or a D. Zeiss; and for examinations for parasites a 1-12 oil-immersion lens for use with a condenser. The eye-pieces used are generally Nos. 2 and 4.

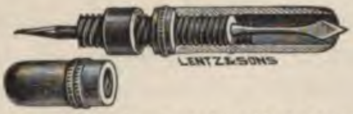
Perfectly clean cover-glasses and slides, such as are used in ordinary microscopic work, are also needed. It is of the greatest importance that these glasses shall not only be free from dust and dirt, but also that they shall not be finger-marked. After ordinary washing they should be washed in ether and alcohol, and then carefully dried over an alcohol flame, or wrapped in tissue paper until needed for use, or kept in 50 per cent. alcohol. The operator should also use a pair of fine forceps provided with a clip, so that he can pick up a cover-glass without touching it with his fingers, and lay the glass down without releasing it from the forceps. (See Fig. 157.)

FIG. 157.



Stewart's forceps for holding cover-glass.

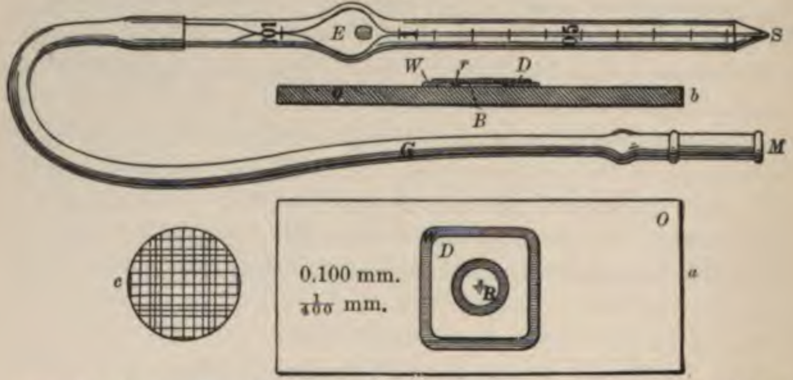
FIG. 158.



Lance for stabbing skin of tip of finger or lobe of ear to obtain drop of blood.

The tip of the finger or the lobe of the ear is now washed clean very gently, so as not to cause an artificial hyperæmia, and the skin of the part is then punctured by a tenotome or a small spear-point made for the purpose. (See Fig. 158.) The first drop of blood which escapes is wiped off, and the surface of a cover-glass is touched to the second drop, so that the centre is wet with the blood. It is then placed with wet side down on a clean glass slide, which has been slightly warmed, using just enough pressure to make an even film of blood between the glass surfaces, and examined under the microscope. This gives so crude an idea of the true state and number of the corpuscles, that it is usual to employ more delicate technique according to the object sought by the physician. If he wishes to know the number of the red cells, he resorts to what is called a hematocytometer, of which the best is that called the Thoma-Zeiss apparatus. This is composed of two parts. One part is a glass capillary tube, about 10 cm. long, with an expansion near the middle, which expansion contains a small glass ball, which is movable. On the tube are three marks. Part way up it is marked 0.5, below the expansion 1, and above the expansion 101. The second piece of apparatus is a

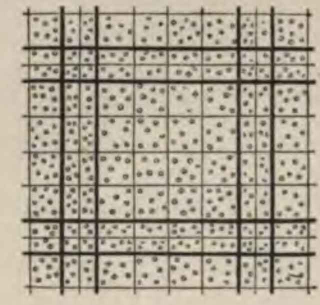
FIG. 159.



Thoma-Zeiss blood-counting apparatus. A heavy glass slip (*a*), in the middle of which is a cell (*B*) exactly $^1/_{10}$ millimetre in depth. The cell is limited at the periphery by a circular gutter to prevent fluid placed upon the cell from flowing beyond it between the slip and cover-glass. The floor of the cell is ruled into squares whose sides are $^1/_{20}$ mm. Dark lines mark out large squares containing twenty-five small squares. Thick, carefully ground cover-glasses (*D*) are provided in the case. The ordinary Potain *Melangeur* (*S*) is used to measure and mix the blood. It consists of a capillary tube the upper portion of which is blown into a chamber (*E*) holding 100 c.mm. The stem of the tube is graduated at 0.5 and 1 c.mm.

somewhat heavy glass slide, upon which is cemented a square of glass. In the centre of this square is placed by the maker a small disc of glass, which is not as thick as that forming the surrounding square, and the surface of it is ruled in 400 minute squares, and these squares are separated into groups of 10 by plainer lines. (See Fig. 160.) If a thin cover-glass is now placed over the square, so that it rests on the edges of it, it will not touch the surface of the centre disc, but leave the space of 0.1 millimetre. Looking from above downward there is seen a cover-glass, and under it a disc which it does not touch; next the disc or around it is a little moat between it and the square of glass. It is upon the upper surface of the ruled disc that the drop of diluted blood, about to be described, is placed.

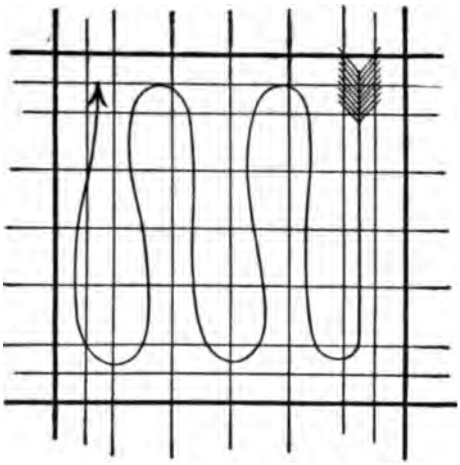
FIG. 160.



Appearance of blood in the Thoma-Zeiss cell.

The finger or ear having been freshly pricked and the first drop wiped off, the blood is drawn up to the mark 1.0 in the capillary tube, and the tip of the tube is then wiped clean. A 0.6 per cent. solution of common salt is drawn up after it, until the tube and bulb are filled to the point marked 101. The tip of the tube is now wiped dry by means of a clean cloth. By shaking the glass ball in the tube the blood and salt solution become well mixed in the proportion of 1 to 100, or if the blood has been drawn only to the mark 0.5, then the dilution is 1 to 200. After the salt solution in the lower part of the capillary tube has been forced out by gently blowing into the upper end of the pipette, and the blood-mixture has reached its tip, a drop of this homogeneous fluid is forced out so that it rests on the surface of the disc, but not so freely that it runs over into the moat about the disc. A cover-glass is now allowed to fall upon the drop in such a way that all air is excluded, and the drop distributed over the surface of the disc. The cell should now be allowed to stand for several minutes to allow the corpuscles to settle and become stationary, and care should be exercised that the stage is perfectly level, as otherwise the blood-cells will all gravitate to one margin. As we look through the microscope, we now see a space of 16 squares surrounded by double lines, and if to these 16 squares we add the squares over which the heavy lines run, we have 36 squares under observation. The corpuscles are now counted in series, following the wavy line shown in Fig. 161, so that the same square may not be counted twice. After all the cells in this group of 36 squares are counted, 5 other groups of 36 are counted. The number of red cells is now added together, and divided by 6, to find the average in each group of 36 squares. This in turn is divided by 36, to determine the number in 1 square, and this number is multiplied by 800,000 if the blood has been drawn up to 0.5 in the pipette, or 400,000 if it has been drawn up to 1. These figures, 800,000 or 400,000, are not arbitrary, but are based on the fact that each square equals 1 : 4000 of a millimetre, and the blood has been diluted 200 times or 100 times. Thus we have the following approximate process as an illustration:

FIG. 161.



Method of counting red cells following the direction indicated, to avoid mistakes.

Total corpuscles in 216 squares = 1296 ÷ 6 = 216 ÷ 36 = 6 × 800,000 = 4,800,000 in a cubic millimetre.

Or if the blood has only been diluted 100 times, then we have the following illustration:

Total corpuscles in 216 squares $= 2592 \div 6 = 432 \div 36 = 12 \times 400{,}000 = 4{,}800{,}000$ in a cubic millimetre.

In making the count, it will be found that some of the corpuscles overlap the line of a given square, and lead to error by being counted twice or left out altogether. For this reason it is customary to include those corpuscles which overlap the upper and left-hand borders. Further, it is best to put down the number of cells found in each square as they are counted, and not to attempt to carry the addition in the memory, since the loss of one corpuscle makes a great difference in the ultimate result, and for this reason the more squares included in the original count the more accurate the result. Immediately after using the apparatus the glass slide should be washed in pure water, not alcohol, and the pipette washed out first with distilled water and then with alcohol, and finally with ether.

In making the count of the red blood-corpuscles care should also be taken to estimate the white corpuscles, since the proportion of white to red cells often gives us very valuable information in disease. This may be done by using as a diluent for the blood in the pipette what is called Toison's solution instead of the solution of salt already named. This has the advantage that it stains the white cells blue, and so renders them more readily counted. Toison's solution is composed of:

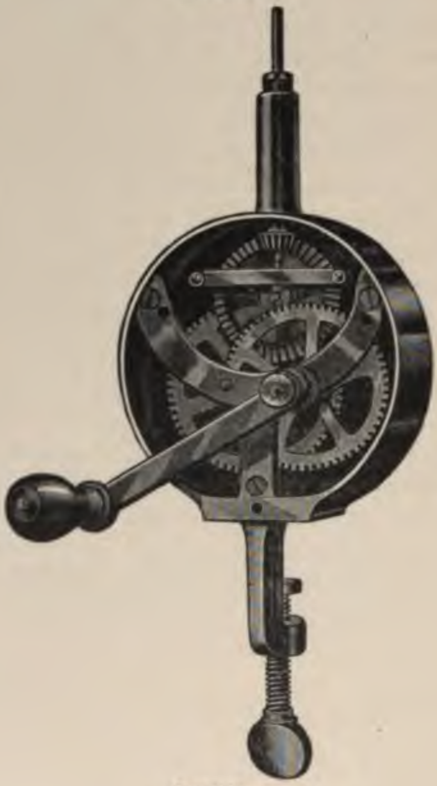
Methyl-violet	0.03	(½ grain)
Neutral glycerin	30.0	(1 ounce)
Distilled water	80.0	(2½ ounces)
Mix thoroughly and add		
Chloride of sodium	1.0	(15 grains)
Sulphate of sodium	8.0	(2 drachms)
Distilled water	80.0	(2½ ounces)

This is then filtered; it requires about eleven minutes for the white corpuscles to be stained by it.

As the red cells are counted the number of the white cells can also be noted in the 400 squares of the field. By counting several fields and taking the average of each the true ratio is obtained. Thus if the average is 8, add three ciphers, and 8000 is the number of white cells present.

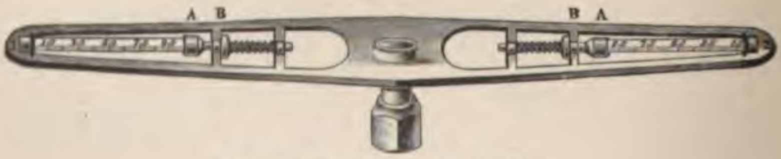
When we desire to count the white corpuscles alone, we employ a pipette (sometimes called the "white pipette") which makes the dilution of the blood in the proportion of 1 to 10, and we use in place of salt solution as a diluent a 0.3 per cent. solution of glacial acetic acid in water. This acid solution dissolves the red corpuscles, but makes the white ones more readily seen. The method of calculating the number of white corpuscles in a cubic millimetre is the same as that given for the red corpuscles, except that as the dilution

FIG. 162.



Centrifuge.

FIG. 163.



Hæmatocrit attachment for centrifuge.

is 1 to 10, instead of 1 to 100, we multiply by 40,000, instead of 400,000.

When the large white pipette is used, it will be found that the blood is sucked up into it much more readily than into the "red pipette," because the calibre of this tube is larger. Greater care must therefore be used that the blood or fluid does not quickly pass up above the marks on its surface. This fact also causes the blood and fluid to run out of the tube very easily if it is held vertically. For this reason the bottle holding the diluting fluid should be tipped a little, so as to keep the tube as nearly level as possible. Because of the large calibre, a greater amount of blood is needed than is usually required for blood examination, and therefore the puncture of the skin must be large enough to permit a free flow.

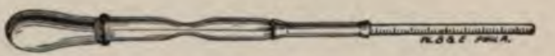
Several fields of white cells should be counted, because the number of white cells found in a single field are too few to enable us to get accurate results, for a variation of one or two cells may make a great difference when they are multiplied by so large a figure. This is the reason that we do not use the red-cell diluting pipette for this purpose. Nearly one hundred white cells should be counted, to obtain correct results.

Another method of rapidly obtaining an approximate estimate of the number of the red and white blood-cells is by the use of the centrifuge, with the hæmatocrit attachment (Figs. 162, 163).

This instrument is used for the volumetric estimation of the red and white blood-corpuscles without previous dilution of the blood. The hæmatocrit attachment consists of a metallic frame, carrying two graduated capillary glass tubes, 50 mm. long, $\frac{1}{2}$ mm. bore, in which is placed the freshly drawn blood. These accurately graduated glass tubes, seated in rubber-cushioned cups at 1 and 2, are held in position securely by spring cups, A, A, so that there is no possible danger of losing the tubes during rotation. By drawing back the milled heads, B, B, the tubes are instantly released and as quickly clamped again into position. (Fig. 163.) This apparatus should be made of aluminum, in order that it may be strong and light. The advantage gained by the use of this metal is that it is possible to greatly increase the length of the arms of the hæmatocrit, thereby taking advantage of the well-known law of mechanics that "the centrifugal forces of two equal bodies, moving with equal velocity at different distances from the centre, are inversely as their distance from the centre." In order, therefore, to obtain any desired amount of centrifugal force it is not necessary to increase the speed of the machine, but simply to increase the distance from the centre, or, in the case of the centrifuge, the length of the hæmatocrit.

The finger of the patient is thoroughly cleansed with water, and then punctured by means of a spear-pointed needle. The first drop of blood is rejected, and a second drop is secured by very slight pressure. The blood is then drawn, by suction, by means of a constricted dropper (Fig. 164) with rubber-bulb connection, into the capillary tube, which is then placed in the hæmatocrit, which is rapidly revolved for at least one minute.

FIG. 164.



The rapidity and simplicity of this process are apparent at once. The blood does not have time to coagulate, and by the centrifugal force the red corpuscles, having the greatest specific gravity, are thrown to the distal extremity of the tube, and will occupy about one-half of the tube, or to about the mark 50.

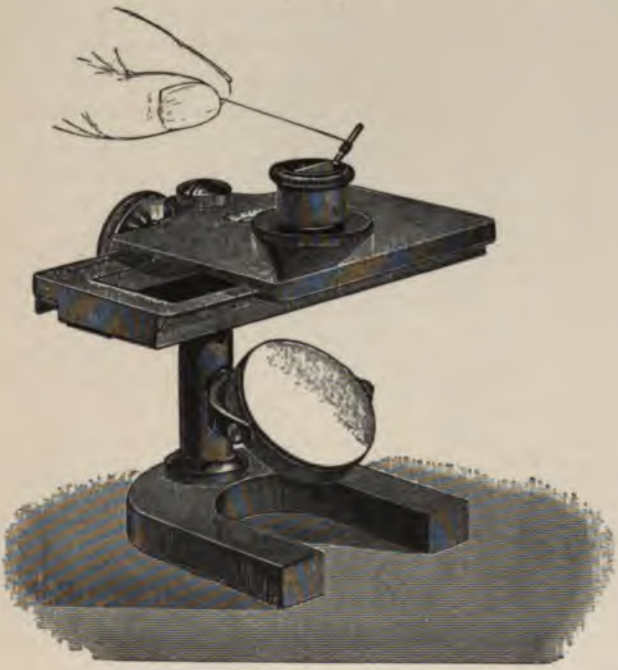
The white corpuscles, next in specific gravity, will occupy a position between the red corpuscles and the liquor sanguinis, which is found in the proximal end of the tube, quite clear and free from corpuscles.

When the column of red blood-corpuscles extends to mark 50 we have, as a rule, about five million red corpuscles per cubic millimetre; but if the precipitated corpuscles reach only mark 30, there are only about three million per cubic millimetre, or sixty volume per cent. If they reach only mark 20, there are about two million per cubic millimetre, or forty volume per cent.

While this method of blood estimation was highly thought of when first introduced, it has now largely lost popularity, and is not commonly relied upon, because it is subject to grave errors.

Having discovered the number and the quality of the corpuscles, an equally important measure is to discover the quantity of hæmoglobin which they contain. This is often done by the use of the hæmoglobinometer of v. Fleischl, although that of Gowers is sometimes used. Fleischl's apparatus consists of a small table, in the centre of which is a hole into which fits a round cylinder with a glass bottom, divided perpendicularly in the middle by a metal diaphragm, and both sides of which are filled with pure water. Under the stand is a frame in which is set a piece of colored glass as near the hue of diluted blood as possible, and this glass is tapered off gradually, so as to give a lighter shade of red at one end than at the other. The frame carrying the glass is marked by a graduated scale, and is moved from side to side, in a track under the half of the cylinder which is to contain only pure water, by a thumb-screw. Under the glass and cylinder is a white reflector to direct the rays of light through them. (Fig. 165.) The rest of the apparatus consists in a little capillary tube attached to a tiny wire handle. This tube will hold just enough pure and healthy blood to color the water on one side of the cylinder to the hue shown in the colored glass when it is opposite the normal mark "100." The finger being punctured, the end of the capillary tube is lightly touched to the drop of blood, which runs up the capillary tube. The blood is

FIG. 165.



Von Fleischl's hæmoglobinometer.

now washed from the capillary tube into one of the sections of the hæmometer container, by directing through the tube a stream of distilled water from a fine-pointed pipette. Both compartments are then filled to the brim with distilled water. The inside of the capillary tube should be perfectly dry before filling it with blood, and, when filled, no blood must be allowed to cling to the outside. Neglect of these precautions invites serious inaccuracies in the results obtained. The apparatus is then exposed to candle- or lamplight, because with daylight the hue of the glass does not match blood-color, and the frame is moved backward under the side of the cylinder which contains only pure water, until the glass seems to the eye to match the fluid containing the blood on the side through which the pure light streams. If the glass matches the blood-color when the mark on the frame is at 50, it shows that the hæmoglobin equals only 50 per cent. of normal; or if it is at 85, it signifies 85 per cent. As a matter of fact, an examination of perfectly healthy blood will often give not more than 85 to 90 per cent. of hæmoglobin with this apparatus.

Care should be taken in regard to three points: first, to be sure that all the blood is washed out of the capillary tube into the water; second, to be sure that the two halves of the cylinder are filled to the brim with water, so that there is neither a positive nor a negative meniscus; and, third, to be careful to cleanse the entire apparatus thoroughly after each use of it before putting it away.

A better apparatus than that of von Fleischl has recently been introduced by Dr. Arthur Dare, one of the assistants in my clinic at the Jefferson Medical College Hospital. It may be described as follows:

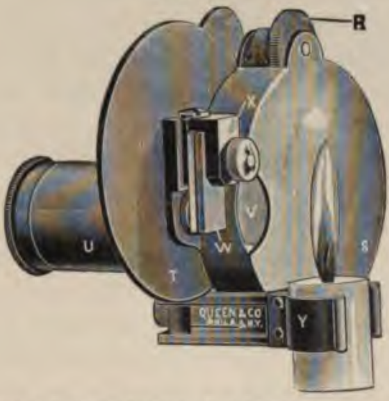
The application of this instrument depends upon the principle that the color of a thin film of undiluted blood, illuminated by candle-light, may be compared with a graduated color comparison.

It has the advantage over others employed for a similar purpose, that while but a small drop of fluid is needed for its application, this consists of pure blood undiluted with artificial serums.

The technique and possible errors of dilution are thereby entirely eliminated, and leucocytosis, which is a frequent source of error in hæmoglobin estimations, imparting as it does a turbidity to mixtures of blood and water, becomes imperceptible when viewed by transmitted light against an opaque background, the red color of the hæmoglobin only becoming visible, while the opacity due to increase in the number of leucocytes is incalculable.

Furthermore, the shades of color to be compared are very decided, even in blood markedly deficient in hæmoglobin, and thus facilitates accurate comparison. Besides the colorimetric principle, there is an element of density, due to concentration of coloring-matter present in the blood film, that magnifies the shade of color, giving a wider range between decimal points, insuring more uniform comparison and greatly reducing the "personal equation" existing in different individuals.

FIG. 166.



Dare's hæmoglobinometer. W. Blood holder. R. Wheel to turn color glass. V. Color glass. U. Observation tube. T. Shade. Y. Candle.

The examination, which consists of collecting the blood, and the estimation of hæmoglobin percentage by comparison with the graduated color-scale, requires very little time for its accomplishment.

The plane surfaces of the blood pipette are quickly cleansed and the whole procedure completed in the time required to take the temperature. By reason of this rapid method hæmoglobin estimations are placed among the possibilities of clinical routine.

The pipettes all contain a blood stratum of standard thickness and can be used with any instrument, a distinct advantage over the calibrated pipettes, which have a capacity only adapted to one particular apparatus.

Still another method of estimating the hæmoglobin, which does not require any costly apparatus, is the so-called specific gravity method.

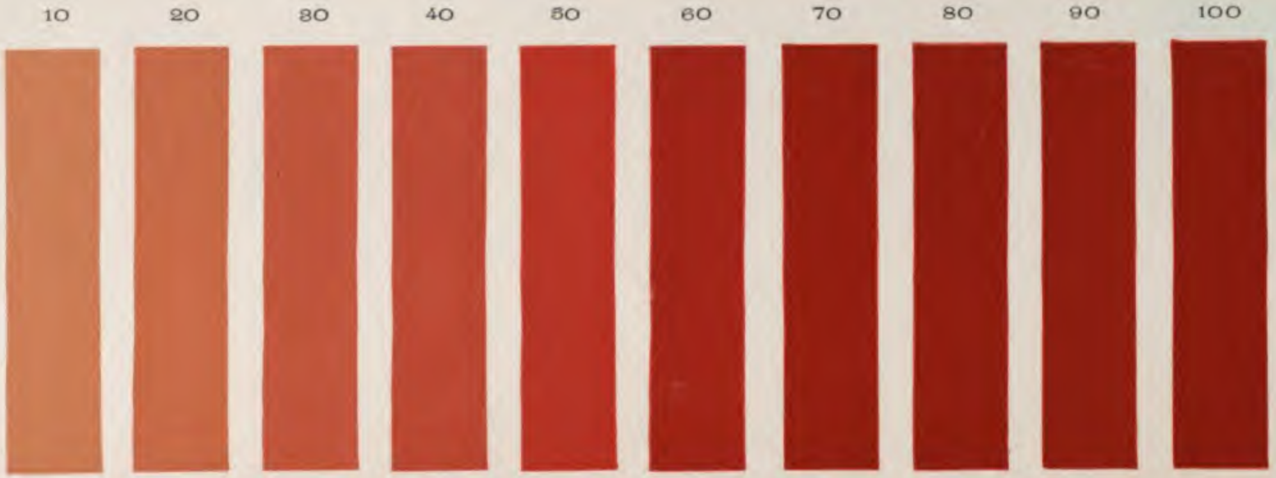
This is Hammerschlag's modification of Roy's method. Chloroform and benzole are mixed in an ordinary urinometer tube, so that their specific gravity is 1.059—in other words, that of normal blood. A drop of blood obtained by the ordinary method of puncture is dropped into this mixture in such a way that it does not touch the sides of the urinometer tube. The drop floats undissolved in the chloroform and benzole mixture. If it sinks to the bottom, its specific gravity is higher than normal, and we add a few drops of the heavier ingredient, chloroform, till the blood drop floats free in the mixture. If, on the other hand, it rises, we add the light benzole till it becomes suspended. The specific gravity is now taken, and if it is above normal we have reason to believe that the hæmoglobin is above normal, or if it is below normal, that the hæmoglobin is scanty. This is true in persons in ordinary disease, but in dropsy, when the red cells may be water-soaked and heavy, or in leukæmia, when the white cells are present in excess, the specific gravity may be excessive and yet the hæmoglobin below normal. Otherwise the test is fairly accurate, since the specific gravity of the blood-cells depends largely on their content of hæmoglobin. The various specific gravities equal the percentage of hæmoglobin in the following proportions, according to Hammerschlag:

1033 to 1035 = 25 to 30 per cent.
1035 to 1038 = 30 to 35 " "
1038 to 1040 = 35 to 40 " "
1040 to 1045 = 40 to 45 " "
1045 to 1048 = 45 to 55 " "

1048 to 1050 = 55 to 65 per cent.
1050 to 1053 = 65 to 70 " "
1053 to 1055 = 70 to 75 " "
1055 to 1057 = 75 to 85 " "
1057 to 1060 = 85 to 95 " "

Not only is it desirable for us to estimate the quantity of hæmoglobin which may be present in the blood, but also to know the relative richness of hæmoglobin in each corpuscle. Thus, in chlorosis we may have red cells present which are nearly normal in number but sadly lacking in hæmoglobin. In pernicious anæmia the reverse condition is present. To determine this, we divide the percentage of hæmoglobin by the percentage of red cells. For example, suppose we find that the hæmoglobin is present in the normal amount of 100 per cent., and a blood-count reveals in a man 5,000,000 corpuscles to a cubic millimetre, we know that the percentage of red cells is also 100. We therefore divide 100 per cent. of hæmoglobin into 100 per cent. of red cells, and 1 is the result. This is said to be the "*color index*," and

PLATE XVI.



Tallquist's color scale for estimating Hemoglobin.

indicates that each cell is normally rich in coloring-matter. The color index may, however, be apparently normal, and yet anæmia be present, for if the red cells be reduced to 2,500,000, or 50 per cent., and the hæmoglobin to 50 per cent., or one-half, the index would be 1. In chlorosis, however, the cells might be 4,000,000, or 80 per cent., and the hæmoglobin 50 per cent., in which case we follow the rule to divide the percentage of hæmoglobin by the percentage of red cells, which would be 50 ÷ 80 = 0.6 +, or if in pernicious anæmia the red cells are 1,000,000, or 20 per cent., and the hæmoglobin is 40 per cent., we find that 80 ÷ 20 = 4. Each cell here contains four times its normal amount of hæmoglobin, yet there is actually a great lack of it.

Another method, not so accurate, but useful, has been described by Tallquist, and consists in taking up a drop of pure blood with pure white bibulous paper, and comparing the color with a correct hæmoglobin color scale. This is, of course, a very simple method. (Plate XVI.)

Proceeding, then, to the examination of the blood for definite diagnostic purposes, we find that a very important part of this study consists in the differentiation of the various forms of white blood-cells. These cells appear in the blood of healthy individuals in five forms:

1. They occur as white cells as small as, or smaller than, the ordinary red cells. Each of these small white cells contains a nucleus so large that it almost completely fills the body of the cell and prevents us from seeing any surrounding protoplasm. They are called lymphocytes. They form about 20 per cent. of the total number of white cells in health. They are increased in number after food, and diminished by starvation. They are not phagocytic, nor do they exhibit amœboid movement.

2. They occur as what are called large mononuclear leucocytes, cells, much larger than the red cells, possessing a moderately large single nucleus, which is surrounded by a zone of pale, non-granular protoplasm. Sometimes these cells show a change in the shape of the nucleus, and are then called (3) transitional leucocytes. These mononuclear cells make about 10 per cent. of the white blood-cells.

4. They occur as large, white cells with a nucleus of irregular shape (polymorphous nucleus), or a nucleus split up into several smaller nuclei (polynuclear). They are often called for this reason

polymorphonuclear or polynuclear leucocytes. Their protoplasm contains fine granules, which stain when brought in contact with neutral dyes, and for this reason these cells are often called neutrophiles. A neutrophile, a polymorphous leucocyte, and a polynuclear leucocyte are, therefore, one and the same thing. They equal about 60 per cent. of the white blood-cells; are actively amœboid and phagocytic.

5. We find very large polymorphonuclear cells containing very coarse granules, which stain when brought in contact with acid dyes, of which the chief is eosin. They are called eosinophilic, and are very limited in number or entirely absent in many cases. These cells possess amœboid movement, but are not phagocytes. They make up in some individuals about 2 per cent. of the leucocytes. When they are increased this indicates leukæmia or trichiniasis.

In disease we find variations from these types as to proportional and actual number, and in addition other white cells are present.

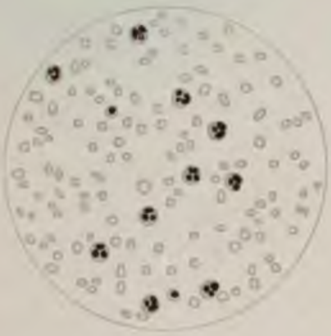
In leukæmia of the lymphatic type the lymphocytes are the cells greatly increased.

In order that the various forms of white cells that we have named may be readily separated from one another we have to resort to certain stains, it having been shown by Ehrlich and many others that the nuclei of these cells are susceptible to different stains, as are also the granules found in their protoplasm. These stains differ as to their color and reaction. We have basic stains and acid stains, but it is not to be thought that these terms mean that they are actually acid or basic in their chemical reactions. These terms are largely arbitrary. As a rule, the acid stains affect the protoplasm of a cell, and the basic stains the nuclei. The basic stains are methyl-blue, methyl-violet, methyl-green, hæmatoxylin, and gentian-violet. The acid stains are picric acid, eosine, and acid fuchsin. The cells whose nuclei stain with basic stains are sometimes called basophiles, those whose protoplasm is stained by acid stains acidophiles (eosinophiles), and those cells which stain with both acid and basic stains neutrophiles. Further, these stains render the nucleus one color and the granules another, as in Plate XVII., in several of the figures of which will be found cells with red granules and blue nuclei.

The best solution for staining purposes is that of Ehrlich, which is called a triple stain. It is composed as follows: saturated watery solution of orange "g," 6 c.c.; saturated hydro-alcoholic (20 per cent. of alcohol) solution of acid fuchsin, 4 c.c. These ingredients having been mixed gradually and thoroughly shaken, the following constitu-

PLATE XVII.

FIG. 1.

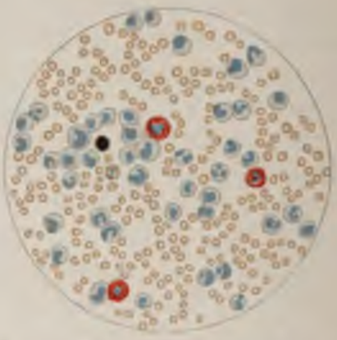


Severe Anemia with Leucocytosis.

Dry preparation. Fixed with picric acid. Stained with hæmatoxylin Böhmer, x 300.

Red corpuscles few, almost colorless, varying in size, show poikilocytosis; two nucleated reds (normoblasts). The increase in the white cells seen to be in the polynuclear elements. (Rieder's "*Atlas der Klinischen Mikroskopie des Blutes.*")

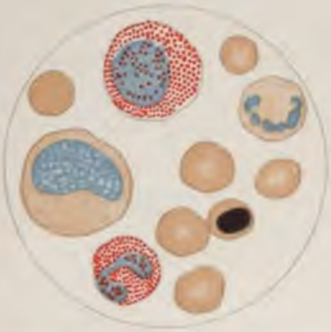
FIG. 2.



Splenic-Myelogenic Leukæmia.

Eosin-hæmatoxylin, x 300. Red corpuscles rosy-red, of nearly uniform size, round. To the left a normoblast with eccentrically placed nucleus. Many large mononuclear leucocytes (myelocytes) and three eosinophiles seen. (Rieder.)

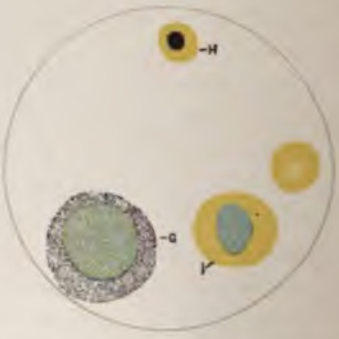
FIG. 3.



Splenic-Myelogenic Leukæmia.

Same case. Eosin-hæmatoxylin, x 1100. One normoblast, one polynuclear leucocyte, one myelocyte, two eosinophiles. The neutrophilic granules of the polynuclear leucocyte and of the myelocyte do not show with this stain. The large mononuclear eosinophile above is believed to be also a myelocyte (Markzelle), the smaller one below, an eosinophile such as can be found in normal blood. (Rieder.)

FIG. 4.



Myelocyte, Normoblast, Megaloblast.

Triple stain. G, myelocyte showing neutrophilic granules; H, normoblast, both from a case of splenic-myelogenic leukæmia; I, large nucleated red corpuscle (megaloblast) from a case of pernicious anemia. (Osler.)

ents are added, the shaking being continued: saturated watery solution of methyl-green, 6.6 c.c.; absolute alcohol, 10 c.c.; glycerin, 5 c.c.; water, 5 c.c., and shake well. This solution should stand for twenty-four hours, to allow of sedimentation; it improves with age; and when it is used the supernatant liquid is to be drawn off by a pipette in order to avoid the sediment. It must not be filtered. This stain acts in a few minutes.

Cover-glasses having first been well cleansed with alcohol and water, the surface of one is touched to a drop of freshly drawn blood, and then another cover-glass placed, not pressed, on its surface until the blood is evenly distributed. The glasses are then quickly separated and dried over an alcohol flame or on a hot stage made of sheet copper, and kept at 212° F. for fifteen minutes. After this they are placed in the staining-fluid for from one to four minutes, then washed in pure water, dried, and mounted in Canada balsam or cedar oil. The Canada balsam should not be prepared with chloroform, as it will decolorize the specimen. The glass is then ready for microscopic examination with a one-twelfth oil-immersion lens. The eosinophile granules in the corpuscles will be stained a reddish hue, the neutrophile granules purple, and the nuclei bluish green or blue. (Plate XVII.)

Anæmia. Having studied the methods of examining the blood, we come next to the consideration of the diagnostic value of the conditions which we find in it. We find, first, that anæmia, or blood deficiency, is represented by two conditions, in one of which the pallor and other symptoms are due to a diminution in the number of red blood-corpuscles, while in the other there is a decrease in the amount of hæmoglobin in each corpuscle. In regard to the white corpuscles we find even more valuable diagnostic data, since their variation in number, form, and character is marked in some diseases. Practically all conditions of the blood which are pathological represent diseases in organs connected with the blood directly or indirectly, and do not depend upon primary changes in this liquid, except in rare instances.

A patient's blood having been found lacking in the proper number of red blood-corpuscles, the question naturally arises as to what conditions underlie this variation from the normal. The most common causes of this decrease are the infectious diseases, which all result in producing a degree of anæmia most marked during early convalescence, and the history of such an attack should always be sought for, and, if found, regarded as an important point for consideration in reaching a diagnosis. If there be no history of acute illness, the most natural condition to be thought of is that known as simple anæmia, produced by no apparent disease of the organs of the body, but due to lack of good food, pure air, proper hygienic surroundings, and exercise. If this is excluded from the diagnosis, we must not forget that if food is taken and not absorbed properly the corpuscular richness of the blood is decreased, and, therefore, chronic indigestion, notably that condition called atrophy of the gastric tubules, may be the cause of the difficulty. Again, profound anæmia, as to the number of the red blood-corpuscles, may be present and seem inexplicable, until it is discovered that the patient suffers from bleeding hemorrhoids, and the daily loss of blood, even though it be small, is sufficient to produce anæmia. Similarly repeated attacks of nose-bleed or of excessive menstruation may so result. Naturally the physician will have excluded the possibility of the anæmia being due to a profuse hemorrhage from any cause before searching as far as this for a diagnosis.

There still remains to be considered the anæmia which is called *pernicious*, in that it progressively gets worse until death occurs in the majority of cases, although a few may recover. At present we do not understand the pathology of this disease. It is characterized by marked pallor without loss of flesh, or, to speak more correctly, the subcutaneous tissues are added to rather than robbed of fat. There are gradually increasing dyspnœa, failure of strength, cardiac palpitation, venous murmurs, some vertigo, and roaring in the ears. The blood shows a most extraordinary and continually diminishing number of red blood-corpuscles, until the number may amount to only 143,000 to the cubic millimetre. In addition, the following points of great diagnostic importance are to be noted: First, the individual red corpuscles are richer than normal in hæmoglobin; second, many of them are larger than normal (megalocytes); third, the red corpuscles are deformed, some being ovoid, others irregular in shape from projections and constrictions on their surfaces (poikilocytes); fourth, there are present microcytes, or red blood-cells which are smaller than normal; fifth, nucleated red blood-cells (normoblasts); and sixth, and quite constantly, there are other large cells like the megalocytes, named megaloblasts, which have a pale staining nucleus. These last are often larger than the megalocytes, and are sometimes called the "corpuscles of Ehrlich,"[1] since he regards them as pathognomonic of pernicious anæmia. The white blood-corpuscles are normal in number, or slightly decreased, although the great diminution in the red cells renders the proportion of white to red greater than normal.

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[1] These are not to be confused with the myelocytes of Ehrlich, which are large white cells.

Anæmia depending upon lack of hæmoglobin in the corpuscles, rather than a decrease in their actual number, is seen most typically in that condition called *chlorosis*. In this state the corpuscular diminution is so slight that it may be ignored; but the decrease in hæmoglobin is extraordinary, sometimes falling as low as 20 per cent. of the normal or below it. The red corpuscles are, however, very commonly irregular in form—that is, there is more or less poikilocytosis, but the white corpuscles remain normal in number or slightly increase. Normoblasts are occasionally found in chlorosis of a severe type, but the larger varieties of nucleated erythrocytes are seldom seen. The diagnostic points, in addition to those of chlorosis just named, are the facts that the patient is generally a girl of from fourteen to twenty-five years, that the skin is peculiar in its pallor (see chapter on Skin), and that there is often little if any menstrual flow, which is usually only faintly pink in hue. Dyspnœa, cardiac irregularity, constipation, and a wayward appetite are often present. Auscultation of the neck on the right side over the jugular vein will reveal a peculiar murmur called a "humming-top" murmur. Febrile movement of slight degree may also be present.

In addition to these causes of anæmia we find anæmia due to a decrease in both the corpuscles and hæmoglobin. A large proportion of these cases have already been mentioned when speaking of the anæmias of convalescence and hemorrhage, but a far more important cause of this condition, yet one often overlooked, to the great regret of the physician in later years, is the possibility of the cause being tuberculosis. Still other causes of such anæmia are cancer, sarcoma, and renal disease, particularly gastric cancer, in which condition the blood may resemble that of pernicious anæmia, and gastric ulcer, in which the loss of corpuscles may also be extraordinary, even if no hemorrhage occurs. Chronic lead-poisoning, arsenical poisoning, and uræmic poisoning may cause it, and it arises from the presence of numerous forms of parasites in the bowels, such as tapeworm, anchylostomum duodenale, and last, but by no means least, from malarial infection, either as manifested by acute attacks, frequently repeated, or by slow poisoning with the development of cachexia. (See further on in this chapter.)

Estimations of hæmoglobin and red cells are of value in certain acute conditions. If a patient is brought to the physician in a state of advanced anæmia, acute or chronic, and needs an operative procedure for relief, and the hæmoglobin is below 35 per cent., it is well to remember the rule of Mikulicz, which is, "Do not operate."

Again, a great decrease in hæmoglobin and red cells would aid in separating the collapse of concealed hemorrhage, as in a case of rupture of the tube in extra-uterine pregnancy, from other conditions of collapse not dependent on loss of blood.

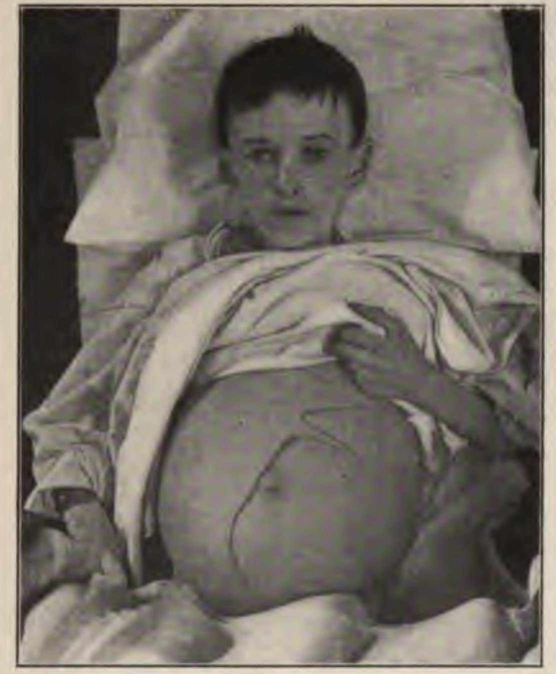
It is claimed by Justus that the diagnosis of syphilis may be made by studying the state of the hæmoglobin before and after the use of mercury. That is, if the index of hæmoglobin is at 80, and mercury is given in a single dose by inunction or hypodermically, the hæmoglobin will fall to 70 or 60, and then rise to a spot higher than before. This is said not to occur in non-syphilitic persons, but the test is open to grave errors.

There yet remain to be considered those conditions in which we find not only the states already described, but, in addition, marked alterations in the white blood-corpuscles as well as the red, alterations of such moment that they become the salient features of the blood when it is examined. These are of great diagnostic importance.

The points of importance in examining blood in regard to the white corpuscles are their number and their peculiarities and kinds. Before doing so, however, it must be borne in mind that the proportion of white cells in different individuals varies very greatly in the limits of health, being increased by the taking of food (digestion leucocytosis), by the condition of pregnancy (gravid leucocytosis), by violent exercise, and by massage. Starvation correspondingly decreases the white cells. Digestion leucocytosis usually lasts for three to five hours after a meal. In these changes the relative percentage of all forms of white cell is increased except the eosinophiles, although in some cases the polymorphonuclear cells are increased in excess. Again, much depends upon the general state of the patient's health and nutrition. A shrivelled, old woman may have as low a proportion as 40 per cent., and a stalwart, athletic man have 80 per cent. and be in perfect health. Usually it may be considered that about 60 per cent. of these polymorphonuclear cells is normal, but the individual characteristics must be taken into account in determining the diagnostic value of the percentage.

When the disease leukæmia is present in its spleno-medullary form, the typical change in the blood is slight increase in the large mononuclear leucocytes and in the presence of the so-called myelocyte of Ehrlich, a large white corpuscle with a single pale-staining nucleus of even outline, usually placed to one side, and neutrophilic fine granules. The myelocyte is frequently of the same size as the large mononuclear leucocyte, and can be distinguished from this cell only by the fact that it contains neutrophilic granules, while the protoplasm of the mononuclear leucocyte is clear. The ordinary large mononuclear cells are not greatly increased, and the polymorphous or polynuclear cells are rather decreased. The myelocyte of Ehrlich is a giant-cell, without amœboid movement. Eosinophile cells may or may not be present. At one time they were supposed to be pathognomonic of this disease, but they are not so considered at this time. When stained with Ehrlich's tri-acid stain the granules of the eosinophilic leucocyte appear as coarse reddish-bronze gran-

Fig. 167.



Spleno-medullary leukæmia, showing outline of enormous spleen in a boy of fourteen years. (Author's wards.) (See also Fig. 151.)

ules scattered through the protoplasm of the cell, and often superimposed upon the nucleus.

The other changes found in leukæmia are that the red blood-cells are greatly decreased in number, and a large number of nucleated red cells may be seen. The hæmoglobin is also decreased. The proportion of one to three red cells is often met with. The symptoms of this form of leukæmia are pallor and puffiness of the face, dyspnœa, and general feebleness, with great and gradual enlargement of the spleen and liver, and marked splenic tenderness. Auscultation over this organ may reveal a murmur and palpation a crepitus. Hemorrhage, generally from the nose, is common, and dyspnœa and diarrhœa are often present. Frequently retinitis develops, and slight fever may occur. This is by far the more common form of leukæmia.

In advanced anæmia, when the proportion of white to red cells is one to ten, the leucocytes being normal in number, except the lymphocytes (that is, the mononuclear, deeper-staining cells, with a rim of non-granular protoplasm), which are greatly increased in number, we suspect *lymphatic leukæmia.* Myelocytes, so typical of the splenomedullary form, do not appear in this condition, and splenic enlargement is absent, but in its place there is often enlargement of the superficial lymph-glands, but these never grow so large as in Hodgkin's disease, or pseudo-leukæmia. (See chapter on Fevers.)

Pseudo-leukæmia, or Hodgkin's disease, is to be differentiated from true leukæmia by the blood examination, it being stated that in this malady there is usually only a slight decrease in the red cells and no other marked changes. Recently, however, Martin and Matthewson have emphasized the fact that in some cases of pseudo-leukæmia the lymphocytes have increased as greatly as in many instances of true leukæmia. As a general rule, however, the blood condition separates the affections. (For Banti's Disease, see page 357.)

We study the white cells as to their number and character for other diagnostic purposes, namely, for the discovery and separation of acute infectious diseases. Thus, in nearly all infections associated with acute inflammation we find an increase in the white cells, or leucocytosis. The particular white cell increased is the polymorphonuclear cell, and rarely we find eosinophiles. In trichinosis the leucocytosis is chiefly connected with eosinophiles. Acute croupous pneumonia is particularly apt to produce this state unless the infection is very malignant, when it fails to occur. By studying leucocytosis we can often decide as to the presence of deep-seated inflammations. Thus in doubtful cases of appendicitis a study of the leucocytes will aid the diagnosis and separate the condition from obscure non-inflammatory states. If an increase of the polymorphonuclear cells is present, we can exclude gallstone colic, renal colic, fecal colic, and ovarian neuralgia, unless there is associated with one of these affections an acute inflammation. The degree of leucocytosis in a case of appendicitis in the early stages is in direct ratio to the severity of the process. If it is absent after several days' illness with appendicular symptoms, its absence may indicate sepsis of a very grave form. So, too, if leucocytosis gradually increases, it is a sign of spreading inflammation. It is important to recall in this connection the fact that a high leucocyte count is met with very early in intestinal obstruction. If it speedily falls, intestinal gangrene is to be suspected. Leucocytosis is usually absent in typhoid fever, malaria, severe septicæmia, tuberculosis in all its forms, in influenza, and in measles; but should some complicating focus of acute inflammation be developed, then it appears. In suspected typhoid fever the absence of leucocytosis, with a diminution of the multinuclear cells and an increase in the mononuclear cells, is of importance in pointing to the presence of this malady. Should perforation of the intestine ensue, leucocytosis is apt to occur, unless a general peritonitis is produced, when the number of leucocytes in the peripheral circulation may be decreased by the outpouring of cells into the abdominal cavity.

A moderate leucocytosis occurs after child-birth, and is normal at this time.

Toward the end of prolonged fatal illness, or where the onset of death is gradual, an increase in white cells develops, called terminal leucocytosis.

Parasites of the Blood. We still have for consideration the parasitic diseases of the blood. These consist in the malarial germ of Laveran, or, as it is more properly called, the "hæmatozoon malariæ" of Marchiafava and Celli, and the filaria sanguinis hominis.

MALARIAL ORGANISMS. No more important addition to the study of disease from a diagnostic stand-point has been made than the discovery of the presence of a parasite in the blood of persons suffering from malarial fever, a parasite which is always present under these circumstances, and in all probability acts as the cause of all malarial manifestations. These parasites are varieties of sporozoa, which live inside the red blood-corpuscles of the individual attacked.[1]

The parasite of malarial fever occurs in three forms, namely, as that of tertian fever, that of quartan fever, and as the parasite of the so-called æstivo-autumnal fever. The tertian parasite is a small hyaline, colorless body, which occupies but a slight extent of the interior of the red blood-corpuscles. (Plate XVIII., Figs. 2, 3, and 4.)[2] When quiet it is round, like the corpuscle in which it lies; but if the specimen examined be fresh, it may be seen to possess active amœboid movements, thereby changing its shape.

Soon this amœboid body grows in size and begins to develop reddish-brown pigment-granules (Plate XVIII., Figs. 5, 6, and 7) in itself. These pigment-granules are rapidly moving bodies, and, as they are often found in the projections of the parasite, it may look, until this fact is corrected by fine focussing, as if several parasites were in one corpuscle. As the pigment-masses increase, the corpuscle which contains the parasite becomes more and more pale, and at the same time swells up or expands, and the amœboid movements grow less and less, while the pigment tends to arrange itself toward the periphery. (See Plate XVIII., Figs. 7 and 8.) Finally, only a shell of corpuscle is left (Plate XVIII., Fig. 9), the pigment after collecting in the centre becomes motionless, and then the parasite undergoes segmentation; and, finally, we have developed 10 to 20 segments, arranged about the central clump of pigment like a rosette. Each segment has a spot looking like a nucleus, and soon the mature bodies so formed break out of their host and attack new and previously healthy blood-cells. Sometimes the parasite becomes so large that it entirely destroys the corpuscle and floats free in the blood, in which case the pigment-granules quiet down and the mass becomes misshapen and apparently dead, breaking up into smaller masses, and gives rise to several small bodies, which, however, soon seem to lose life (Plate XVIII., Fig. 21), or it becomes filled with vacuoles (Plate XVIII., Figs. 23 and 24), or, finally, we have springing from these extra-cellular bodies flagella or waving arms, extending from the margin of the parasite. (Plate XVIII., Fig. 33.) These flagella break off now and again and keep waving through

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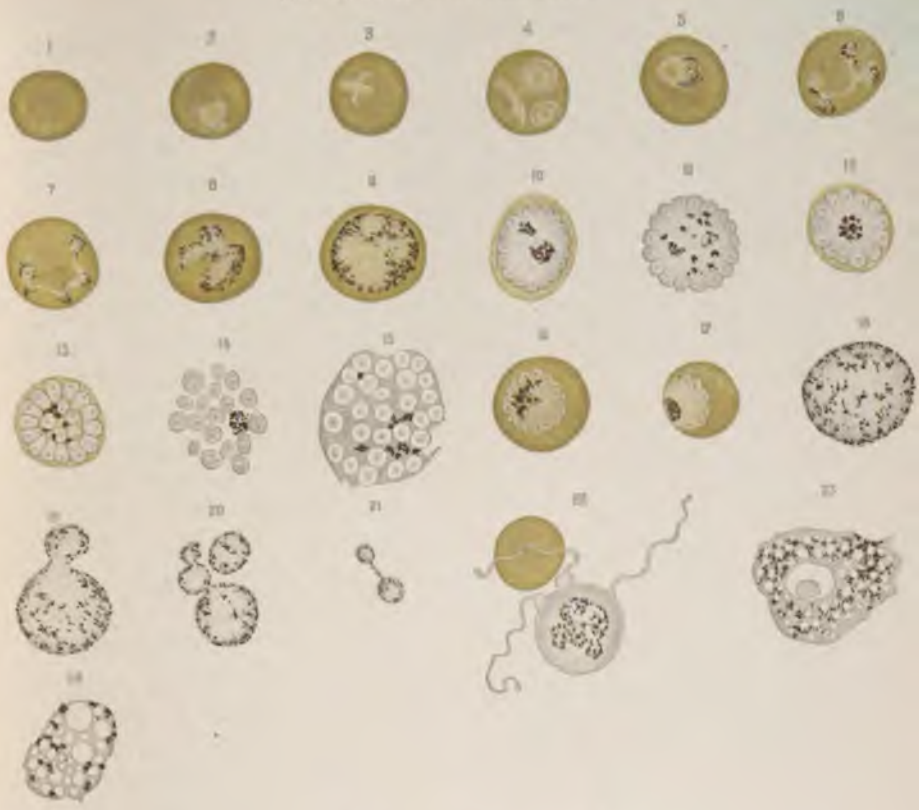
[1] In this country the chief investigators into the life-history of the malarial parasites have been Osler, Councilman, and, more recently, Thayer and Hewetson, from whose exhaustive and able monograph of "The Malarial Fevers of Baltimore" much of the information in the text of this book is derived.

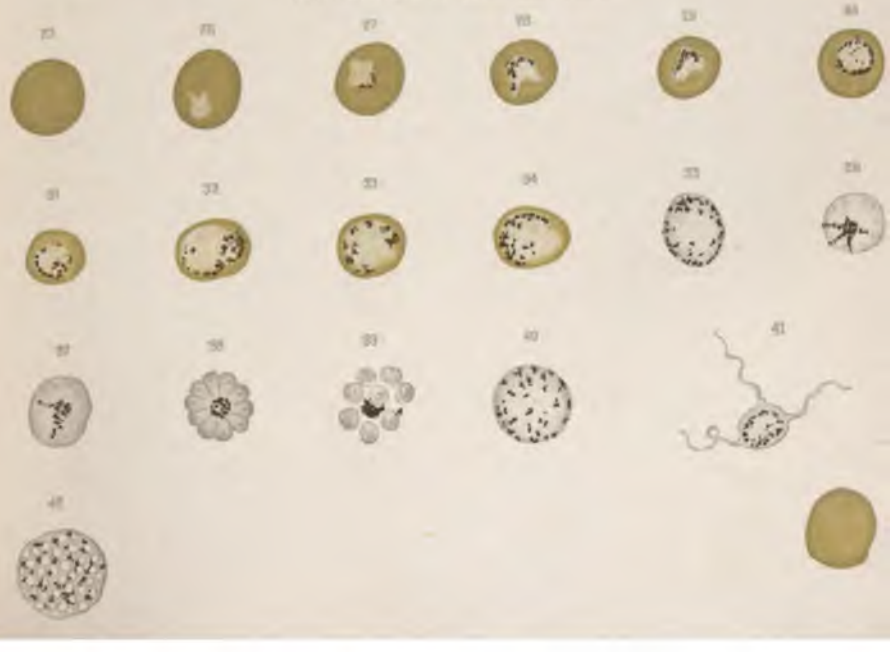
[2] No. 1 is a normal red corpuscle. Plates XVIII. and XIX. are taken from Thayer and Hewetson's monograph.

PLATE XVIII.

The Parasite of Tertian Fever



The Parasite of Quartan Fever



the blood, looking like spirilla. The entire process just described seems to consume about forty-eight hours, and it is of interest to note that the acme of the paroxysm of the disease occurs with the segmentation of the full-grown parasite, so that the presence of segmenting bodies indicates the near approach of an attack. If, on the other hand, we have a double tertian infection—that is, an attack daily—or a quotidian form, we have two sets of parasites, each one of which reaches its period of segmentation on alternate days, and so a daily attack is caused. In such blood during a paroxysm will be found two sets of parasites: one set segmenting or causing the paroxysm, and the other set half-developed, which will produce the attack of the morrow.

The quartan parasite, or the one causing an attack every third day, in its earlier stages of development looks very much like that of the tertian form, for it occurs as a small hyaline amœboid body filling a fraction of the corpuscle. It soon, however, develops the following differences: first, it develops a sharper outline; second, it is more refractive; third, the amœboid movements are slower (Plate XVIII., Fig. 26); fourth, the pigment-granules are coarser and darker (Plate XVIII., Fig. 27), and, more important still, they lie very quietly around the edge of the parasite; fifth, the corpuscle acting as host does not increase in size, and finally disappear, as it does when affected by the tertian type, but grows smaller and darker, more refractive and metallic looking. (Plate XVIII., Figs. 28 to 34.) Reaching its complete development in about sixty-four to seventy-two hours, it appears as a small round body, taking up nearly all the space in the corpuscle in which it lives, or it appears free in the blood-serum. (Plate XVIII., Fig. 35.) As the time for the paroxysm approaches, the pigment-granules which have been scattered begin to collect at the centre (Plate XVIII., Figs. 36 to 39) in a stellate form, and the protoplasm of the mass then divides by segmentation into from six to twelve small pear-like bodies, each of which has a refractive centre. These bodies become more and more separated from one another, and simultaneously we find new corpuscles infected by the original small round bodies which we first saw.

Sometimes these parasites expand and become very transparent, their pigment-granules become very active, but finally become quiet, and the body of the parasite grows more and more indistinct. They become dead parasites. (Plate XVIII., Fig. 40.)

Again, the parasites may undergo a breaking up into smaller bodies, which are badly formed and indistinct; a degenerative form may also appear, and vacuoles may develop. (Plate XVIII., Fig. 42.) Finally, flagella may develop, as in the tertian organism (Plate XVIII., Fig. 41), and they differ from the tertian form in being smaller, and their granules are coarser.

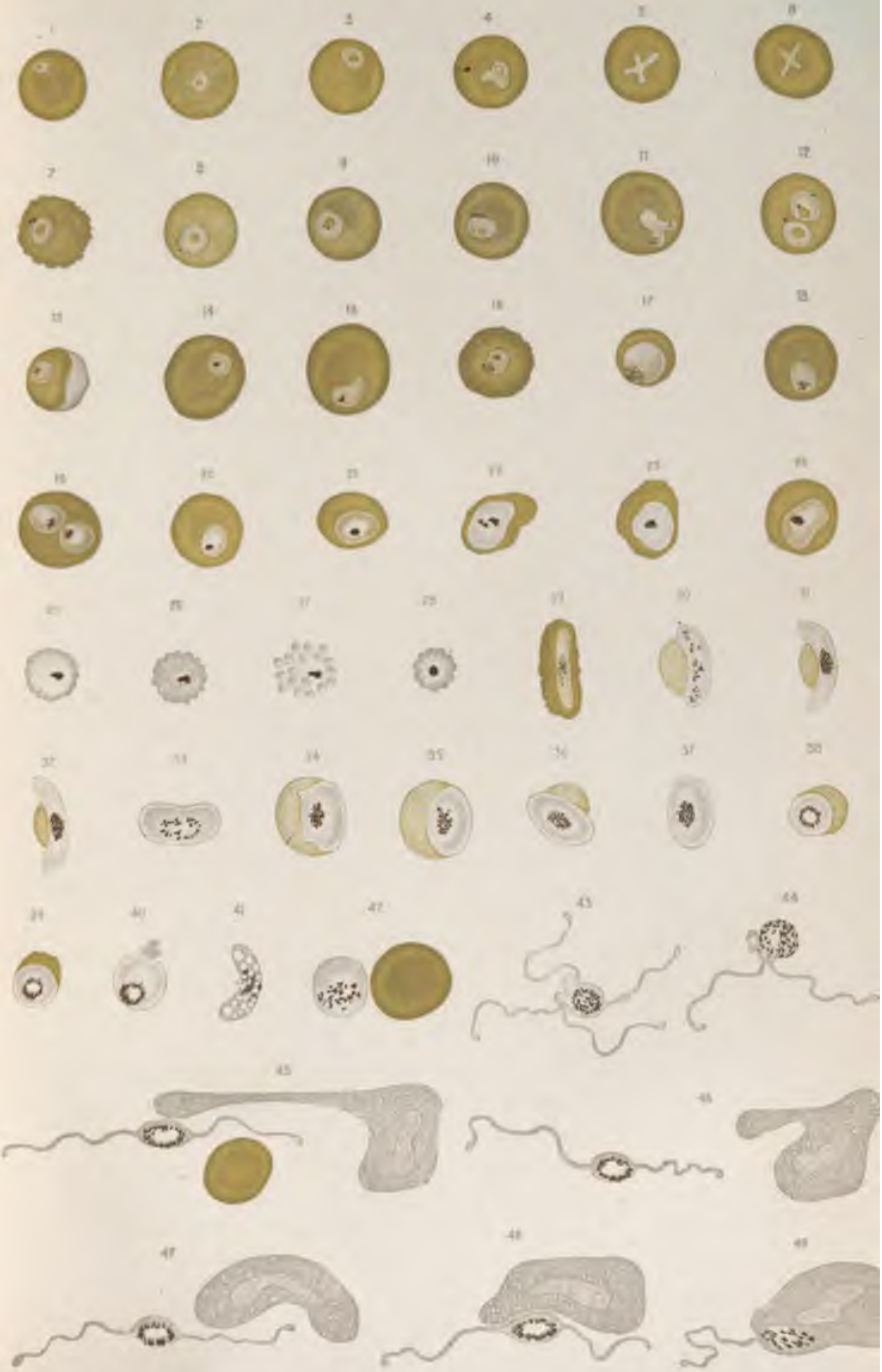
In the third form of infection (æstivo-autumnal fever) we find at first the small hyaline bodies, but they have a ringed appearance, and are sometimes very small. (Plate XIX., Figs. 3 to 6.) Suddenly this body becomes larger and the ring is lost, the edge becoming wavy, and amœboid movements occur, the pseudopodia, often joining to form a true ring. Pigment-granules finally develop after a variable length of time, but they are few, rarely more than two in a parasite, are near the edge (Plate XIX., Figs. 7 to 12), and quite still. The corpuscles are not decolorized, but often are shrivelled and very brassy looking.

The peripheral circulation during the paroxysm of æstivo-autumnal fever contains very few, if any, parasites, but blood drawn from the spleen may show intracorpuscular parasites, with blocks of pigment and some free parasites. As segmentation goes on the parasite may look like the tertian form, but it is far smaller. (Plate XIX., Figs. 21 to 28.) After this parasite has been present for some days we find in the blood larger parasites, of an egg-shape or crescent-shape, the remains of the blood-cell, looking like a "small quarter of an apple glued to the side of the crescent," with central clumps of pigment-granules. It is these bodies which are characteristic of æstivo-autumnal fever. (Plate XIX., Fig. 29.) Vacuolization and flagellation may develop in this form as in others, and the use of quinine in the first week may prevent the development of the crescents.

There is a great difference in the ease with which the various forms of the malarial parasite are found in the blood in the peripheral circulation. In infection with the tertian parasite, during the later stages of development the organism is much more frequently found in the blood of the spleen than in the peripheral circulation. In cases of quartan infection, the parasites appear in the peripheral blood as well as in the spleen, and in the internal organs in all stages of development; while in the æstivo-autumnal form of the disease, only the young forms appear in the peripheral circulation, and when the parasites are arranged in groups no organisms may be found in

PLATE XIX

The Parasite of Aestivo-Autumnal Fever.



the peripheral circulation during the paroxysm. This may also hold true in regard to the quartan and tertian parasites, and the entire cycle of development may take place in the internal organs, so that examination of the peripheral blood may reveal very little. In other words, while the discovery of the parasite in a suspected case confirms the diagnosis, the absence of the parasite does not absolutely negative the opinion that it is the cause of the malady. Of course, a very considerable number of slides should be carefully examined before it is stated that malarial parasites cannot be found. Given a case in which a considerable number of parasites are found in the peripheral circulation, we have a right to infer that the infection is severe. But this does not necessarily follow. In examining the blood for the æstivo-autumnal parasite, it will often be found that the best results are obtained if the blood is stained. For while, as a rule, the examination of a fresh specimen is the best method of examining the blood for the malarial parasite, it is a fact that it is difficult to discover the small hyaline forms in a fresh specimen. Under these circumstances Thayer recommends Nocht's modification of Romanovsky's stain. This method is as follows:

Polychrome methylene-blue is carefully neutralized. This is done by first adding dilute acetic acid until the solution is acid. When litmus paper is dipped into the solution, it is colored by the methylene-blue, but on the margin of the moist portion the acidity causes a red line to appear. The solution is now brought back to the neutral point by the addition of more polychrome methylene-blue until the red line fails to appear on the blue litmus paper. The polychrome methylene-blue may be obtained already prepared. It can, however, be readily made by heating for several hours, on a water-bath, a solution consisting of:

Methylene-blue	1 part.
Caustic soda	1 part.
Water (distilled)	100 parts.

After cooling, the solution is to be filtered.

To this polychrome methylene-blue an equal quantity of distilled water is added, and then a saturated solution of ordinary methylene-blue until the red color is completely lost, the solution appearing simply blue. For this purpose, about 1 part of the saturated aqueous solution to 10 parts of the diluted polychrome solution will be used.

A solution of eosin is now prepared, according to Nocht, by adding

3 or 4 drops of a 1 per cent. aqueous solution of eosin to 1 or 2 c.c. of water. This is practically a 0.2 per cent. solution; it can be made up in quantity. To 3 or 4 c.c. of the eosin solution, Nocht adds the methylene-blue solution until the red color disappears. At this point a fine precipitate is thrown down, and a scum begins to form on the surface.

Thayer also advises that the eosin and methylene-blue be mixed immediately before being used, and the mixture used only once, since when it is kept a day or two, it gives the chromatin stain, although feebly. The best method of fixing the blood-smears for this stain is to treat them with 0.25 per cent. solution of formalin in 95 per cent. alcohol for one or two minutes. When this stain is used, the hyaline forms of the parasites appear as delicate blue rings, the central part being occupied by a large colorless nucleus, at one side of which is a small spot of chromatin substance, which stains a deep carmine-violet.

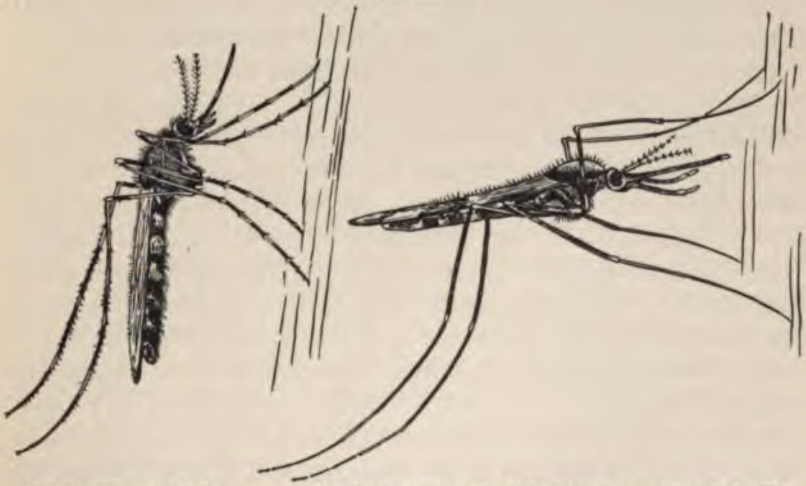
The following table separates each of these malarial forms from the others:

Tertian Parasite.	*Quartan Parasite.*	*Æstivo-autumnal.*
Develops in 48 hours.	Develops in 72 hours.	Develops in 24 to 48 hours.
Pale and indistinct.	Sharply outlined and refractive.	Has a winged appearance.
Actively amœboid.	Slightly amœboid and later motionless.	Actively amœboid.
Pigment fine.	Pigment coarse.	Pigment-granules are very few.
Pigment active in movement.	Pigment slow in movement.	Pigment-granules quite still.
Pigment light.	Pigment dark.	
Full size of the corpuscle.	Smaller than the corpuscle.	
Degenerative forms twice as large as corpuscle.	Degenerative forms very much smaller than in tertian.	Very much smaller than a corpuscle.
Segments 16 to 20.	Segments equal 6 to 12.	The process of segmentation goes on in the internal organs, so segmenting form is not found in the blood.
Irregular segments often.	Beautiful rosettes.	Forms crescents.
Corpuscle becomes colorless and swollen.	Corpuscle becomes brassy-looking and shrunken.	Corpuscle is shrivelled and very brassy, but not decolorized.

The bodies which are crescentic and those which are ovoid are not capable of sporulation, and are called gametocytes. While they are incapable of development in the human being, in the intermediate host, the mosquito, or even on the slide of the microscope, the micro-gametocytes or male elements give off a number of long motile flagella (micro-gametes), which, breaking loose, penetrate and fecundate the female forms, or the macro-gametes. This fecundated female form now enters the wall of the stomach of the mosquito, where it undergoes development within forty-eight hours; there then develops in the stomach wall of the mosquito small, round, refractive bodies of granular appearance, and which contain pigment-granules. Five days later these bodies develop striæ, due to the development of sporoblasts, and finally the original organism or mother oöcyst bursts, and sets free a multitude of small sporoblasts, which find their way into the veneno-salivary glands of the mosquito, and so get into the body of the patient, where they develop into young parasites. The other shapes than those which are crescentic or ovoid have an asexual development and infect new blood-cells without having to pass into the body of the mosquito. The *Anopheles* is the only mosquito which is known to act as a host in the way described. It occurs in several forms in the United States, the most common being the *Anopheles quadrimaculatus*, which is identical with the *Anopheles claviger* or *maculipennis* of Europe.

This mosquito is to be separated from the other mosquitoes by the following points. The wings are mottled, the posterior pair of legs is nearly parallel to the wall on which the insect rests, although they may be lifted over the back, in the same manner as does the *Culex*. The body, instead of resting parallel with the resting surface, is at an angle of 45 degrees, whereas, the body of the non-malarial mosquito rests parallel with this surface. (Fig. 168.)

FIG. 168.



The picture to the left represents the *Culex* or non-malaria-bearing mosquito at rest. The figure to the right shows the resting attitude of the *Anopheles quadrimaculatus*. (Modified from drawing in the British Medical Journal.)

The blood is usually examined for the malarial parasite by what is called the direct or "without staining" method. The cover-glasses which are to be employed are cleansed very carefully by washing in alcohol and ether. The lobe of the ear, after being carefully cleansed, is then stabbed with a needle or small tenotome, and the first few drops of blood are wiped away. A perfectly clean cover-glass is now picked up by means of a pair of forceps and touched to the tip of the drop of blood and then placed blood-side down upon a clean glass slide. The blood is equally distributed between the glasses, and only the merest touch of the cover-glass should be made to the drop of blood, as otherwise too much blood will be taken up.

The microscope should be fitted with a 1-12 oil-immersion lens and a No. 4 eye-piece.

When it is desired to keep the specimen and to stain it, the best stain is that of methylene-blue and eosine, which is prepared as follows: a concentrated watery solution of methylene-blue is diluted one-half with water and mixed with an equal volume of a ½ per cent. solution of eosine in 60 per cent. alcohol.

In order to prepare blood-specimens for staining, two cover-glasses are required. After a small drop of blood has been allowed to fall on the centre of one cover-glass the second cover-glass is placed upon it, and as soon as the blood has spread out between the two in a thin film the glasses are drawn apart, the surface of each bearing a thin, even layer of corpuscles. The cover-glasses are now rapidly dried in the air, and immersed in a mixture of equal parts of absolute alcohol and ether for two hours, to "fix" the preparations. The specimens are then dried and stained by Löfler's methylene-blue mixture, which consists of concentrated alcoholic solution of methylene-blue, 30 c.c.; solution of caustic potash (1 to 10,000), 100 c.c. The specimens are immersed in this stain for one-half to one minute, then washed in pure water, dried by placing them between two pieces of filter-paper, and then mounted in oil or balsam. The nuclei of the parasite and of the leucocytes will appear blue and the red cells will be unstained.

We can also use another method, in which we stain by placing the dried cover-glass in Chenzynski's solution and gently heating it for fifteen minutes. This solution is made as follows: Methylene-blue in saturated water solution, 40 c.c.; eosine in ½ per cent. solution in 70 per cent. alcohol, 70 c.c.; distilled water, 40 c.c. The hæmatozoa are stained blue, the red cells take the color of eosine, the nuclei of the leucocytes are stained blue, and the eosinophile granules of the cells bright red.

Perhaps the best method of staining for use by the busy general practitioner is that of Futcher. The blood having dried upon a cover-glass, a 1 per cent. solution of formalin in 90 per cent. alcohol is poured upon it in small quantity, and left in contact for one-half to one minute. The specimen is dried between leaves of filter-paper, and then washed with absolute alcohol, after which it is dried a second time, and then stained by immersing it in Marchand's solution of phenol thionin for twenty or thirty seconds. The specimen is then washed in water, dried between filter-paper, and mounted in balsam. The blood-corpuscles stain a pale-green, the parasites a deep-violet hue. If stained too much, the pigmented granules in the parasites may be obscured. Thayer states that this method is particularly useful in bringing out the small ring-shaped bodies of the parasite of æstivo-autumnal fever with their chromatic dot. The formula for Marchand's solution is:

Saturated solution of thionin in 50 per cent. alcohol,	20 parts.
Two per cent. solution of carbolic acid,	100 "

The solution must stand several days before using.

It has already been stated that the paroxysm of the malarial disease takes place at the time when the parasite is breaking up into segments. In other words, the attacks occur whenever the cycle of growth of a set of parasites is completed, which in tertian fever is every forty-eight hours and in quartan fever every seventy-two hours. If there be two sets of parasites in the blood, however, of the tertian type, the attacks may be daily, or quotidian, since each set mature on alternate days. This is often called double tertian. This is the most common form of the disease in the United States. If there be a double quartan infection, the attacks come on two successive days, then a day of intermission ensues. If three sets of parasites of this type are present, the attacks may be daily for three days—triple quartan infection. (See chapter on Fever.)

The parasite of æstivo-autumnal fever is irregular in its development, and is often the cause of the irregular malarial fever seen in the fall of the year. It yields less readily to quinine than others.

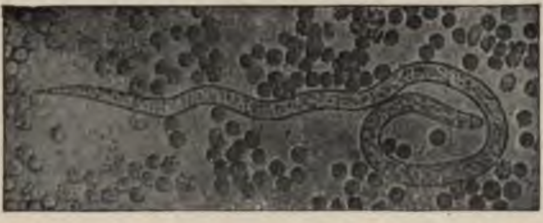
Malarial infection differs from most infections, the symptoms of which resemble it, in that there is no increase in leucocytosis, whereas in sepsis a great increase is usually present. This gives us an important aid in differential diagnosis. (See chapter on Fever.) When the malarial organisms cannot be found the presence of leucocytes bearing pigment granules may indicate the breaking down of the red cells by the parasite, and so point to the probable presence of malaria.

The Widal test varies in the constancy of its development with the stage of the disease, being most marked in the third and fourth weeks. In a large series of cases Park found it present in 20 per cent. of cases in the first week, 60 per cent. in the second week, 80 per cent. in the third week, 90 per cent. in the fourth week, and in 75 per cent. at the end of two months. The only difficulty with this test is that it fails to appear at a time when the diagnosis is often most difficult, namely, in the first few days of the illness.

FILARIA. The filaria sanguinis hominis appears in the blood in its embryonal form, and is found fully developed only in the lymphatics. It occurs in three forms, and has been well described by F. P. Henry, of Philadelphia. These forms are: 1. *Filaria diurna;* 2. *Filaria nocturna;* 3. *Filaria perstans.* These names are indicative of the habits of the animal, the *filaria diurna* being found in the superficial vessels solely or chiefly during the day; the *filaria nocturna* solely or chiefly during the night; while the *filaria perstans* is constantly present in the capillaries of the integument. The *filaria diurna* and *filaria perstans* are confined, thus far, to the west coast of Africa and adjoining districts, while the *filaria nocturna* is pandemic in the tropics and endemic in certain sections of the United States. The adults of *filaria nocturna* have been frequently found; that of *filaria perstans* never, so far as Henry has been able to ascertain. Manson has also described another parasite, the filaria Demarquayi, which is less than half the size of the filaria nocturna, and another form which he calls the filaria Ozzendi. In the opinion of Manson, the *filaria loa* of the eye of the negro of Old Calabar is probably the adult form of the *filaria diurna.* If it is not, he argues, then there must be another bloodworm yet to be discovered, for the embryos of the *loa* must escape from the body of their host through the medium of the circulation. The *filaria perstans* has been practically proved by Manson to be the cause of the fatal "sleeping-sickness" of the Congo region.

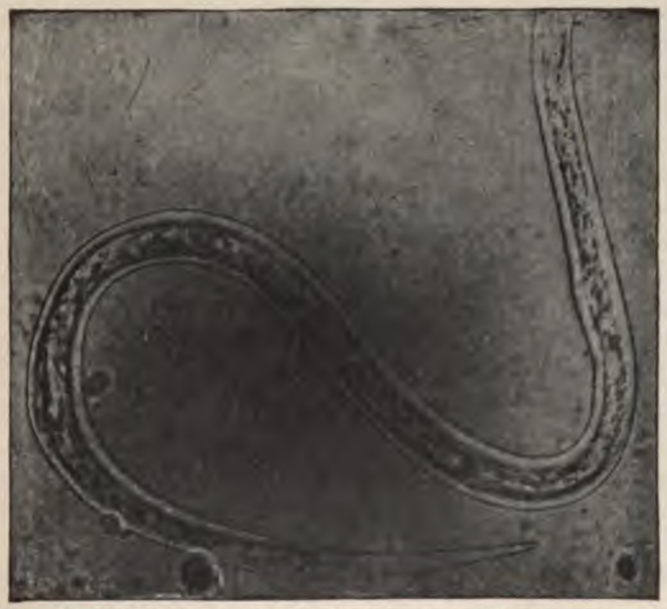
The second is the one ordinarily seen in blood obtained from the peripheral circulation during sleep or at night. (Figs. 169 and 170.) The male filaria measures 83 millimetres long by 0.407 millimetre broad, and the tail is twisted into a spiral form. The female measures 155 millimetres long by 0.715 millimetre wide, and the vulva is 2.56 micromillimetres from the anterior extremity. The embryo measures 270 to 340 micromillimetres long by 7 to 11

FIG. 169.



Filaria alive in the blood. Instantaneous photomicrograph. Four hundred diameters magnification. Four millimetres Zeiss apochromatic. (HENRY'S case.)

FIG. 170.



Filaria in the blood. Eight hundred diameters. (HENRY'S case.)

micromillimetres wide, and has a pointed tail. This embryo is in an almost imperceptible shell, which does not impede its movements, and as it is about the size of a red blood-corpuscle it passes through the capillaries in extraordinary numbers. Its active movements and typical appearance render it readily seen in the blood. The discovery of this parasite in the blood renders a diagnosis certain, and it should always be sought for if chyluria or elephantiasis is present. If the patient remains awake at night and sleeps during the daytime, the organism will be found in the blood during the sleeping-period.

The filaria diurna is found in the blood during waking-hours, and the embryos of the filaria perstans are the only form of this parasite known.

WIDAL'S TEST FOR TYPHOID OR ENTERIC FEVER depends upon the fact that the blood-serum of a patient suffering from typhoid fever exercises an agglutinative effect upon the typhoid bacillus. The method of Widal, as modified by Johnston, of Montreal, is as follows: The lobe of the patient's ear having been pricked, the drop of blood is placed on a clean glass slide and allowed to dry. A loop of bouillon culture of the typhoid bacillus is now placed on an absolutely clean cover-glass, and to this is added a large loopful of a watery solution of the dried blood. From the mixture of blood and typhoid bouillon a "hanging-drop" preparation is made, and examined with a $\frac{1}{6}$ or $\frac{1}{8}$ dry objective, when it will be noticed, if the patient is suffering from typhoid fever, that the typhoid bacilli rapidly form clumps. If the patient has not typhoid fever, this clumping and entanglement of the bacilli with arrest of their movements does not take place, unless he has had the disease within several months, when the reaction may occur without signifying the onset of a new attack. Partial clumping is not to be considered indicative of a positive test.

Sugar in the Blood. The blood in persons suffering from diabetes contains sugar in excess, and R. T. Williamson has suggested its discovery by the following process. The normal blood will not give this reaction. A small, narrow test-tube is well cleaned, and at the bottom of the tube are placed 40 c.mm. of water. To measure this the capillary tube of a Gowers hæmoglobinometer is used, which is graduated for 20 c.mm. The tip of one of the patient's fingers is cleaned and dried, then pricked, and when a large drop of blood has escaped it is sucked up into the small capillary hæmoglobinometer tube. Twenty c.mm. of blood are taken up from the finger. The blood is then blown gently into the water at the bottom of the small test-tube. If it should adhere to the side of the tube, it must be carefully shaken to the bottom. Then 1 c.cm. of a 1 in 6000 watery solution of methyl-blue is added. Finally, to the mixture 40 c.mm. of liquor potassa are added. The contents of the tube are then well mixed by shaking. As a control-experiment a second test-tube of similar size is taken, and into this is placed the same quantity of non-diabetic blood, with the same proportion of water, methyl-blue, and liquor potassa.

The fluid in each tube has a fairly deep-blue color. Both tubes are then placed in a beaker or capsule containing water. This is heated over a spirit-lamp until the water boils; it is allowed to continue boiling for about four minutes. By the end of this time the fluid in the tube containing the diabetic blood has changed its color from fairly deep blue to a dirty pale yellow (almost the color of normal urine), while the fluid in the tube containing the non-diabetic blood remains blue, occasionally it becomes bluish-green, sometimes pale violet, but it is never decolorized—that is, it never loses its blue color. The tubes should be kept quite still while in the water-bath, as by shaking the decolorized methyl-blue is oxidized by the oxygen of the atmosphere, and a blue tint may then return to the fluid. This is the reason why it is necessary to use a water-bath, since if the test-tubes be heated directly over the spirit lamp it is difficult to avoid shaking the fluid.

If the assertions of Freund are correct, the testing for sugar in the blood is of value as a means of separating carcinoma and sarcoma in diagnosis. Thus he asserts that in carcinoma there is an increase in the sugar in the blood, whereas in sarcoma no such increase takes place.

For this purpose the test just named is scarcely delicate enough, and it is wise to remove the proteids by boiling with sodium sulphate, and then after filtering to apply the ordinary urinary tests for sugar.

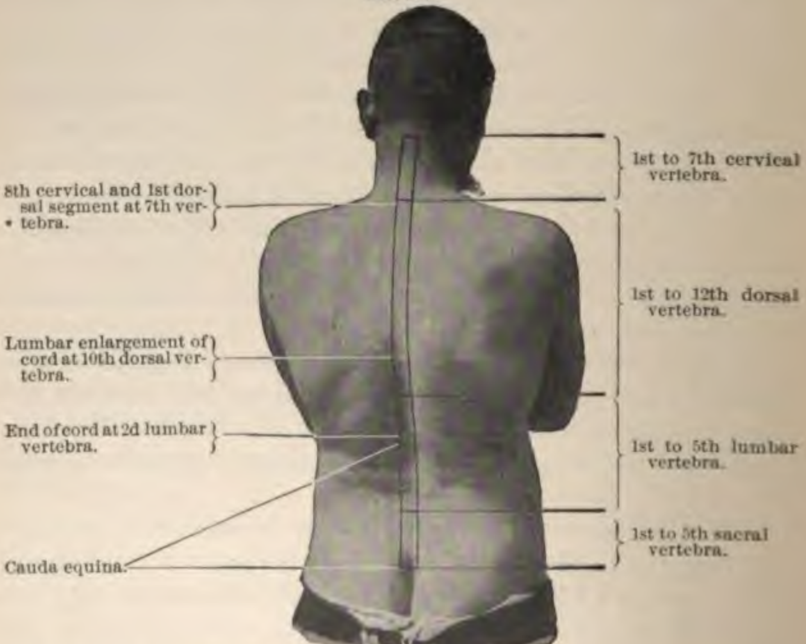
CHAPTER XI.

THE URINARY BLADDER AND THE URINE.

Disorders and diseases of the urinary bladder—Retention of urine—Incontinence of urine—The characteristics of normal and abnormal urine—The normal and abnormal contents of the urine—Their significance—Tests for the contents of the urine.

The Bladder. The objective symptoms of bladder difficulties are generally local, unless they are very chronic, when the face may appear worn and weary, and, if a purulent cystitis be present, septic fever may occur. The subjective symptoms are tenderness, tenesmus, pain (see chapter on Pain and chapter on Abdomen), and retention or incontinence of urine. Retention of urine usually

FIG. 171.


1st to 7th cervical vertebra.
8th cervical and 1st dorsal segment at 7th vertebra.
1st to 12th dorsal vertebra.
Lumbar enlargement of cord at 10th dorsal vertebra.
End of cord at 2d lumbar vertebra.
1st to 5th lumbar vertebra.
1st to 5th sacral vertebra.
Cauda equina.

Showing the surface-areas of the back corresponding approximately to the areas of the spinal cord supplying the trunk, limbs, and bladder.

depends upon causes arising outside this viscus. It may arise from disease or injury which destroys or temporarily impairs the function of the cells in the spinal cord which govern the contraction of the muscles involved in expelling urine from the bladder. These centres are situated at or about the level at which are given off the second, third, and fourth sacral nerves. (Fig. 171. See also page 121.)

Paralysis of the bladder with retention may, therefore, follow severe injuries to the spinal cord, produced by a fall, blows, or other traumatisms, or be due to a myelitis which destroys such centres. (See chapter on Legs and Feet, part on Paraplegia.) The bladder-symptoms seen in myelitis—transverse, traumatic, or otherwise—usually come on in the acute form within a few hours after the sensory and motor disturbances have been noticed by the patient, and either incontinence or retention, or both, may occur.

If, however, the myelitis is not complete, the bladder may escape. On the other hand, if the portion of the cord which is involved happens to be that part governing the bladder, vesical symptoms may develop before the motor symptoms are clearly marked. Again, it is a noteworthy fact that when recovery takes place vesical control may be regained before any marked improvement can be found elsewhere. Often the loss of control of the bladder is such that the patient cannot voluntarily expel the urine and cannot retain it, and it dribbles away without his knowledge. Under such circumstances there is probably a myelitis involving the lower part of the dorsal cord and the upper and lower parts of the lumbar cord; in other words, all that portion in which the vesical centres are situated. If the dribbling of urine takes place without distention of the bladder, the fluid passing directly from the ureters through the urethra, the lower part of the lumbar enlargement of the cord is affected, owing to the paralysis of the sphincter. On the other hand, distention of the bladder, due to retention of urine, occurs when the myelitis is in the lower dorsal and upper lumbar cord, and is due to paralysis of the detrusor muscles, which make no effort to expel the urine, while the sphincter, the centres of which are intact, maintains a tightly closed orifice. Such cases may empty the bladder spasmodically at long intervals (overflow incontinence) —that is, sphincter-paralysis from distention may ensue. In such a condition the bladder should be emptied by the catheter to avoid paralysis and vesical disease. To put the case in another way, we can say that the spinal centre for the control of the walls of the bladder is situated at a higher point in the cord than is that for control of the sphincter, and, therefore, retention of urine indicates a lesion higher up in the cord than does incontinence without retention. Precisely similar vesical symptoms occur in cases of spinal tumor producing transverse lesions of the cord (see chapter on Feet and Legs, Paraplegia), or may arise from spinal apoplexy.

The bladder-symptoms of locomotor ataxia are often quite characteristic, and are to be separated from those of myelitis, spinal tumor, and the vesical troubles due to traumatisms of the cord. The disorder depends entirely upon interference with the reflexes of the viscus, and so presents varying symptoms which are motor and sensory. The patient sometimes complains of the fact that he has to strain for a long time before he can start a stream, which, even after it is started, is often jerking or interrupted; or, again, he must sit down and bend over in order to have the aid of his abdominal muscles before he can evacuate the bladder. As a result of this, residual urine in excess is always present, and cystitis or milder degrees of vesical irritability develop. In other instances the desire to urinate comes upon the patient so suddenly and forcibly that the urine is voided before he can, with his impaired gait, reach a place to pass it in a proper manner; on the other hand, it may be retained and can only be removed by a catheter. Still others find that urine escapes on laughing, coughing, or sneezing, owing to lack of complete control of the bladder and its sphincter; or, again, after many attempts to urinate, the patient gives up the effort, only to be humiliated by an involuntary passage of urine immediately after his penis has been withdrawn into his clothes.

These symptoms differ so materially from myelitis as to make a diagnosis as to their cause nearly always possible.

In obscure cases of ataxia the vesical symptoms may aid the diagnosis quite markedly; thus the presence of bladder-symptoms would confirm a diagnosis of ataxia as against pseudo-tabes due to peripheral neuritis. Again, in myelitis the presence of vesical symptoms points to that disease, and excludes from the diagnosis such affections as poliomyelitis and lateral sclerosis, affections in which vesical paralysis rarely, if ever, occurs. Precisely similar vesical symptoms are sometimes seen in cases of general paralysis of the insane, but the delusions of grandeur or melancholia and other characteristic signs of this disease separate it at once from ataxia.

Retention sometimes comes on in locomotor ataxia, in which discase the impulses from the bladder are not recognized, or are perverted, so that the sphincter which closes the bladder does not relax to permit the escape of urine, or the cord or brain fails to recognize that the bladder is full, and so sends no impulse for its relief. Again, retention of urine may arise from paralysis of the muscular part of the vesical walls by pressure produced in severe labor (childbirth). Finally, we see cases in which the bladder cannot be emptied, because its walls have been paralyzed by over-distention with urine.

On the other hand, incontinence results from loss of power in the sphincter, due to injury or disease in the cord at the level of the second, third, and fourth sacral nerves; and this, by the way, is a far more frequent occurrence than is absolute retention. The real condition under these circumstances is that the expelling-muscles and retention-muscles are both paralyzed, so that the urine accumulates in the bladder and then dribbles through the unguarded neck of the bladder. Sometimes, too, this incontinence is caused by the urethra being so insensitive that it fails to recognize the presence of the urine, and so does not send an impulse to the sphincter to tighten its hold. Incontinence also results from excessive reflex irritability of the walls of the bladder, so that the urine no sooner trickles into this viscus than an impulse is sent to the spinal centres which send a motor impulse to the muscles of expulsion. This is often the condition in the nocturnal incontinence of children, for as soon as the child sleeps its will-power over the bladder ceases, and reflex activity is alone in control. Irritating, concentrated urine may pervert the reflexes of the bladder and so cause incontinence.

The sensory disturbances of the bladder will be found discussed in the chapter on Pain, but it is worth noting here that accompanying the symptoms already named as characteristic of locomotor ataxia vesical crises of spasm and pain frequently occur.

When there is pain in the bladder, made worse by the attempted act of micturition, and tenesmus, with darting pain into the urethra, there is probably present a cystitis; but the physician should remember that cystitis may be present with almost no painful manifestations, even when in its acute form. In other cases this condition arises from concentration of urine, which produces irritation of the viscus, such as is seen in cases of acute nephritis or renal congestion. In children this concentration of the urine is the most common cause of nocturnal urinary incontinence.

Involuntary passage of the urine sometimes occurs in idiots, in some cases of insanity, in attacks of apoplexy, or any condition of abnormal unconsciousness, and sometimes in very severe infectious diseases, such, for example, as diphtheria. Oftentimes it results in children from irritation of the foreskin or vagina, or from rectal irritation produced by seat-worms, since all these causes disturb the reflex activity of the spinal centres.

Interference with the passage of urine may also arise from two causes which are surgical in character, namely, stone in the bladder and tumors of the bladder, which are often situated near its neck and so produce obstruction. Finally, in old men, that most commonly met with cause of difficult micturition, enlargement of the prostate, is to be remembered.

Aside from these causes of interference with the passage of urine, we must not forget the possibility of its obstruction by stricture of the urethra, nor should the physician ignore the fact that some persons have "nervous bladders," which will not respond to an effort of the will if another person is near by, although the urine is instantly passed as soon as the patient is alone.

The Urine. Changes from the normal in the urine, as already stated, are determined, first, by its general appearance, quantity, odor, specific gravity; second, by its microscopical appearance; and, third, by its chemical reaction and responses to tests. Any changes in this fluid of an abnormal character are solely symptomatic, and point with more or less distinctness to disorders of bodily metabolism, disease or disorder of the kidneys, ureters, bladder, or urethra, and sometimes of the prostate, testicles, vagina, or uterus.

The urinary secretion is one which is too frequently ignored by the student and physician in studying the diagnosis of disease. In many instances it will, if properly tested, give such positive evidence in regard to obscure affections that a correct diagnosis is at once possible, and in other cases its examination, as a matter of routine, will discover important facts, the existence of which has been unsuspected. Again and again will a diagnosis prove erroneous if the importance of urinary examinations is ignored, and costly errors for the patient and the reputation of the physician ensue.

In asking questions about the character of the urine passed and its quantity, the physician should be sure that the patient clearly understands his questions. Often he is told that much urine is passed, when, in reality, it is really in small amount, but passed often; or that it is blood-red, when simply red from urates and uric acid. In inquiring about its color, we should remember that if large amounts of liquid have been swallowed it will probably be light in hue, or, if small amounts of drink are taken, dark in hue. So, too active exercise in warm weather may produce a somewhat concentrated urine, because much liquid has been lost by the skin in sweat, and the muscular exertion produces large quantities of urinary material indicative of tissue waste. In winter the urinary flow is more apt to be free, because the skin is inactive.

Changes from the normal in the urine depend upon anomalous conditions, involving the organs which secrete, retain, and expel the fluid, and which result in alterations in its quantity, odor, specific gravity, its naked-eye appearance, its microscopical appearance, and, finally, in those changes which are discovered by means of tests which reveal the presence of otherwise unrecognized substances.

The objective symptoms of some cases of disease of the kidneys have already been discussed. (See the chapter on the Skin, part on Œdema and Color of the Skin, and the chapter on the Face, part on Expression.) Aside from these evidences of renal disease no alteration can usually be noted unless it be loss of weight, which may be replaced by a gain if dropsy develops. The subjective symptoms of the patient commonly consist in loss of ambition, malaise, disturbed digestion, and shortness of breath. Rarely there is pain in the lumbar region, unless pyelitis, stone in the kidney or ureter, or perinephritic troubles are present, when pain becomes an important sign.

The urine which is to be tested should always be passed directly into the vessel in which it is brought to the physician, and this bottle should be scrupulously clean; or, if the urine is passed into any other vessel, care must be taken that it is perfectly clean. When it is thought that urethral disease may obscure the investigation a catheter should be passed, all urine in the bladder drawn off, and then the catheter allowed to remain in place, so that the urine will trickle directly from the ureters to the catheter, and so to a receiving vessel. This is very important when the urine is voided involuntarily. If the condition of the bladder is bad, this viscus should be washed out by boric-acid injections, in order to prevent it from contaminating the urine which is to be tested.

The quantity of urine passed by a healthy adult varies from two to four pints in the twenty-four hours, according to the amount of liquid ingested, the freedom of perspiration, and the amount of exercise.

The significance of any great and constant increase in the amount of urine passed in a given case is various. Thus, we find it greatly increased in any disease of the diabetic centre, or of the liver, or pancreas, which results in diabetes mellitus; in diabetes insipidus, in some cases of neurasthenia, and in some cases of hysteria. It is also increased in many cerebral lesions. Hypertrophy of the heart, *particularly if associated with chronic contracted kidney,* causes an increase in the urine; and, therefore, if a patient has to urinate frequently or has to arise at night to empty the bladder, we suspect this trouble if diabetes is excluded. The same result ensues if the heart and kidneys are stimulated to increased effort by the action of drugs, such as digitalis, caffeine, or alcohol. We also find an increase in urinary secretion, without its possessing any grave significance, in convalescence from such diseases as typhoid fever and pneumonia.

The quantity of the urine is diminished in cases in which the heart fails to do its proper amount of work, with resulting stasis of the blood in the kidneys, and whenever any large amount of liquid is taken away from the body, as in diarrhœa. It is also decreased by fevers and by the sweats following febrile movement. Persistent vomiting also has a similar effect. Parenchymatous nephritis, both acute and chronic, greatly diminishes the urine, and in grave, fatal illnesses urinary suppression also takes place.

The odor of freshly passed urine is faint, but characteristic. What is often called a "urine odor" is really due to the development of ammonia in urine which has decomposed. The odor is altered by many drugs and foods, notably by copaiba, turpentine, eucalyptus, valerian, musk, asafœdita, and by asparagus. Diabetic urine possesses a heavy, sweet odor.

The naked-eye examination of the urine often gives very important information, if its clearness, opacity, and color are observed. Its clearness and color are modified by the presence of pigments derived from outside sources, such as the educts of carbolic acid or salicylic acid, of senna or hæmatoxylon, urobilin, and many other substances coming from inside sources, such as blood and bile. Many of these causes may render it opaque, but there are two conditions, above all others, which render the urine cloudy even when freshly passed, namely, cystitis with phosphaturia and chyluria. After urine has stood for some hours it often becomes opaque, because it has undergone chemical changes.

When urine is dark red in color and somewhat opaque the discoloration may be due to blood, hæmoglobin, santonin, rhubarb, senna, logwood, or the presence of an excess of urates. Again, it may be rendered almost black, instead of red, by an excess of biliary coloring-matter.

A black urine is sometimes seen in a case of melanotic cancer, or after the brownish urine produced by carbolic acid or uva ursi has been exposed to the air.

If the color be due to blood or *hæmaturia*, the urine will be of a more or less bright red, according to the freshness of the sample brought to the physician and the seat of the hemorrhage. If the urine has been voided several hours, it will be of a dingy red or smoky hue, and on standing will cause a coffee-ground or reddish sediment of a somewhat flocculent appearance. If, on the other hand, the urine is seen as soon as passed, it may be a bright red or a dingy red, according to the seat of the hemorrhage and the time which has elapsed since the bleeding began; if it has arisen in the kidney or ureter or bladder, and has been gradual, the mixture of blood and urine will have been so intimate that changes in the blood will have taken place, whereas if the hemorrhage has occurred, simultaneously with urination, from the neck of the bladder or the urethra, the blood will be almost unchanged when it escapes from the urethra. The presence of clots in recently passed urine indicates a not very recent hemorrhage, and yet one of such size that the urine could not by dilution completely prevent clotting.

When the blood comes from the kidney some of the possible causes are acute parenchymatous nephritis, resulting from any one of the severe infectious diseases, such as scarlet fever or malarial fever; embolism, resulting from ulcerative or other forms of endocarditis, producing renal infarction, sepsis of the kidney; the ingestion of irritating drugs, such as cantharides or turpentine; and strains of, or blows on, the back, producing rupture or other disorganization of the kidney. All these conditions produce what may be called acute hæmaturia. If the cause be acute nephritis from the presence of an infectious malady, such as scarlet fever, the pain in the loins, the presence of albumin in the urine, and the eruption will render the diagnosis easy.

Hæmaturia due to malarial poisoning may appear with the first malarial paroxysm, of the intermittent type, which the patient has ever had, and at a time when the history of the case renders it certain that a hidden malarial condition could not have previously damaged the renal tissues or those of other organs in the body. In other words, there are cases in which a free hemorrhage from the kidney takes place, by reason of the chill, in much the same manner in which hemorrhage takes place in acute nephritis due to exposure to cold or to irritants. Under these circumstances there may or may not be developed a true organic lesion of the kidney in the sense of permanent disease.

Secondly, we have cases in which bloody urine appears, not in the first malarial paroxysm of the intermittent type, but in association with the later attacks, which may have followed the first either rapidly or slowly. In these cases there may be no further cause for the hemorrhage than excessive congestion, but in all probability the vast majority of such patients present distinct renal changes, which permit such a symptom to develop when the paroxysm asserts itself.

Thirdly, we pass from those cases of bloody urine due to intermittent forms to those due to remittent attacks, which, in many cases, have gradually merged from the first into the remittent. In these patients the process by which a bloody-colored urine is developed may be very complicated, since it may be due to renal disease, functional or organic, or to a true hæmoglobinuria, arising from dissolution of the red blood-cells in the bloodvessels or blood-making organs.

Finally, there is a type of malarial hæmaturia which is produced by the administration of quinine (Karamitsas *et al.*).

All these forms of hæmaturia can be diagnosed by the presence of the malarial germs in the blood (see Blood) and the characteristic malarial symptoms, except that which occurs in persons who have a dyscrasia from old malarial poisoning when no organisms are found.

If the hæmaturia be due to embolic infarction of the kidney, an examination of the heart will probably reveal signs of valvular disease, from which source the embolus will have resulted, or in other cases the physical signs, combined with the history, will show malignant endocarditis with renal sepsis therefrom. Sometimes thrombosis of a renal vein occurs in feeble, wasted infants, and so causes hæmaturia. If embolism is not the cause of the hæmaturia, the history of the ingestion of an irritating drug will be the diagnostic guide, or, if injuries be the cause, a history of traumatism will elucidate the case.

The causes of chronic or persistent hemorrhage from the kidney are chronic hemorrhagic nephritis, cancer of the kidney, calculus in the pelvis of the kidney producing ulceration, injury of the kidney by jarring of a stone, tuberculosis of the kidney, and cystic degeneration.

If the chronic hæmaturia arise from chronic hemorrhagic nephritis, the diagnosis is made by the pallor of the skin, anorexia, nausea, headache, œdema, decreased amount of urine, and albuminuria.

If the cause be renal cancer, the cachexia, pain, and the mixture of pus, blood, and disorganized renal tissue in the urine will render the diagnosis possible. If due to calculus, there may be a previous history of attacks of renal colic or of violent pain in the kidney; and if ulceration of the renal pelvis has occurred, there will be disturbances of the body-temperature, pain in the lumbar area, and pus in the urine. The presence of tubercle bacilli in the urine decides the presence of renal tuberculosis. If cystic degeneration is present, it can only be determined if the cyst is large enough to be felt.

A sudden profuse hemorrhage in the urine, sufficiently large to endanger life, may come from cystic tumor of the kidney.

Blood from the kidney usually possesses the following characteristics: it is well mixed with the urine, and is generally altered in appearance to the naked eye, and under the microscope both as to color and the shape of the corpuscles. The cells and casts which may be present are changed in color by the hæmoglobin which is free in the urine. Again, blood-casts or red blood-corpuscles clinging to casts indicate renal hemorrhage. When the blood comes from the kidney pelvis it may appear in the urine in long, worm-like clots (moulds of the ureter), and their extrusion from the ureter produces symptoms of colic. Under such circumstances there may be alternations of hæmaturia and normal urine, due to the blocking of the ureter on the diseased side by a clot, so that all the urine comes from the healthy kidney.

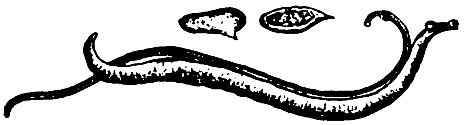
When the blood comes in the first part of the urine passed, and not in the last part, it almost certainly comes from the urethra.

Blood in the urine may not only come from the kidney, but from the bladder, and be due to an acute cystitis, to papilloma, malignant growth, or tuberculosis of this viscus, or to injury. These vesical causes should be excluded from the diagnosis by the use of the cystoscope if possible.

It must not be forgotten also that blood in the urine may be due to menstrual discharge, to blood from uterine fibroids or malignant ulceration, and that it is possible for a malingerer to place blood in the urine, with the object of deception. Rarely in certain cases of locomotor ataxia, hæmaturia develops after the vesical crises which we have already described (see Bladder in this chapter). This is a capillary hemorrhage from the bladder-walls.

There are other varieties of hæmaturia which must not be forgotten, although comparatively rare, namely, that due to the presence in the blood of the filaria sanguinis hominis, which is a condition in which the presence of chyle in the urine so masks that of the blood that the urine has the appearance of pinkish cream or milk, but microscopical examination will show blood-corpuscles and fat-globules, as well as the embryos of the filaria. (See Chyluria in this chapter.) Another still more rare cause of hæmaturia is the distoma hæmatobium of Egypt and Abyssinia. (Fig. 172.) This produces what has been called tropical hæmaturia. The third cause is even more rare in man, namely, the strongylus gigas, which also causes pyelitis and renal colic. A fourth form of hæmaturia is that seen in some cases of scurvy, particularly of the infantile type, and, lastly, hæmaturia may also appear as a symptom of purpura hæmorrhagica, hæmophilia, and very rarely in leukæmia.

FIG. 172.



Distoma hæmatobium, male and female. The two small bodies are the eggs.

The urine, when not discolored by blood, may be discolored by the presence of the coloring-matter of the blood. This is called hæmoglobinuria.

Hæmoglobinuria arises from a number of causes, such as infectious disease, poisoning by mushrooms and poisonous doses of certain coal-tar derivatives, or of chlorate of potassium, or glycerin. Malarial poisoning sometimes causes it instead of hæmaturia. One form of malarial hæmoglobinuria is intermittent, the urine being at one hour limpid, the next hour bloody, and the third hour clear again.

The possibility of confusing the hæmoglobinuria of idiosyncrasy, when in a severe form, with that due to severe malarial poisoning, is very great, for the history of paroxysmal hæmoglobinuria teems with reports of cases in which the chief manifestations of a malarial attack were present, such as chills, fever, and sweats. Probably many cases of so-called malarial hæmoglobinuria are due to another parasite not as yet isolated. Hæmoglobinuria also follows severe burns and the transfusion of human blood, and occurs in paroxysmal hæmoglobinuria, a condition which seems to be produced by mere chilling of the surface of the body or by immersing the hands of a susceptible person in iced water. It may also be produced either by exposure to cold and damp or to the chill of the milder forms of malarial paroxysm. Hæmoglobinuria may also be a symptom of that curious vaso-motor affection called Raynaud's disease.

Microscopical examination of the urine in such cases will show no corpuscles, although the urine will be coagulated by the nitric-acid test; but the coagulum does not settle in flakes, as it usually does in albuminous urine, but floats on the surface in a brownish mass. The naked-eye appearance of the urine is that of clear port wine. If a few drops of this urine be placed on a watch-glass, and a drop of strong acetic acid be added, the blood-crystals of Teichmann will be found by the aid of the microscope, showing that the coloring-matter is hæmoglobin.

If the discoloration of the urine be due to blood, a microscopical examination will reveal red blood-corpuscles, white blood-corpuscles, and perhaps fine filaments of clots; but the corpuscles will not be found in rouleaux, as in ordinary blood outside the body, and they may be crenated and distorted in shape, particularly if the urine is alkaline.

A test which can be most easily applied to determine the presence of blood, if the microscope cannot be used, is Heller's test, which consists in adding to a few c.c. of urine a little caustic soda, so as to render the liquid strongly alkaline. The urine is now heated to boiling, and if blood is present a bottle-green color is produced, and the phosphates fall to the bottom of the test-tube in fine flakes, tinged brownish red by the coloring-matter of the blood.

Still another test, and perhaps the best known to determine the presence of hæmoglobin in the urine, is the so-called guaiacum test. The urine having been placed in a test-tube, a mixture in equal parts of tincture of guaiacum and oil of turpentine, which has been ozonized by exposure to the air, is allowed to flow gently along the side of the tube, so as to rest upon the urine. If blood-pigment is present, a white ring, which gradually becomes blue, forms at the point of contact between the two liquids.

If the urine be red from other causes than blood, this may be due to the ingestion of logwood. The history of the ingestion of this substance will clear up the diagnosis. If it be due to senna, it will be carmine, due to the chrysophan in this drug; but this discoloration only appears if the urine is alkaline. Precisely similar changes are due to the taking of rhubarb. So in santonin-poisoning a blood-red urine is sometimes seen, but it usually attains this appearance after being at first yellow, then saffron, and then purple-red. One of the conditions of the urine, due to a poison, which can be readily confused with hæmoglobinuria or hæmaturia, is that produced by carbolic acid. This color is not due to blood, but to oxidized educts of the acid. The same educts produce a similar discoloration after naphthalin, creosote, and uva ursi have been taken in overdose.

Red urine, due to none of the causes which have been enumerated, may be due to an excess of urates (except urate of sodium, which is usually white). If on the addition of nitric acid the urine becomes brown where the fluids join, the coloration is due to urates; but if all the fluid is brown, the patient has probably been taking freely iodine or compounds of iodine.

Finally, the urine is often dark reddish-brown or porter-colored in jaundice, owing to the presence in it of biliary coloring-matters. Under these circumstances it may be clear or opaque, and the fluid is apt to be frothy on shaking and to have a decreased surface-tension, so that powdered sulphur rapidly sinks to the bottom of the vessel, when the sulphur is dropped on the urine. These biliary colors are at once recognized by the reaction with nitric acid in Gmelin's test, for if a little of the urine be placed on a white plate and nitric acid be allowed to touch the margin of the wet place, a play of colors from green to blue, blue to violet, and violet to red occurs. The green color is the only one characteristic of the biliary reaction, for indican gives with nitric acid the other colors. The same test can be used by wetting bibulous paper with urine, and the acid, if brought to the edge, will stain the paper in the colors named. (For the symptoms of jaundice see the chapter on the Skin.)

A greenish-colored urine is seen in cases of poisoning by salicylic acid, due to the indican and pyrocatechin; and after the use of saffron. Not rarely a greenish or blue urine is due to the ingestion of methylene-blue, either as a medicine, as in the treatment of gonororrhœa, or in candies colored by this dye.

When through disease-processes indican is formed and excreted in the urine, it may by oxidation be transformed into a blue color (indigotin) or into a red hue (indirubin).

Indicanuria is present in intestinal obstruction, general peritonitis, cholera, cancer of the liver or stomach, and pernicious anæmia. It may, however, be present in health as a result of constipation. Blue urine is also caused, as intimated above, by the ingestion of methyl-violet as a drug. (See part of this chapter on Chemical Tests.)

If chromogen is present in large amount, shaking the urine with air will develop a violet-blue color, or this change may take place in the bladder. If urine containing indican be treated with two or three times its volume of hydrochloric acid, it will turn a violet hue.

White or milky-looking urine is seen in that condition called chyluria, due to the presence of the filaria sanguinis hominis in the blood. This urine on standing forms a creamy layer on its surface, and, if it is shaken with ether, some of the fat can be removed, rendering the urine clear. The diagnosis can only be confused by urine becoming mixed with milk or cream, and can always be made if the embryos of the filaria can be found in the urine. They lie in very delicate sheaths, and show a constant vibratory movement. The diagnosis is still further confirmed if they are found in the blood, where they are present in large numbers at night. (See chapter on the Blood.)

Urine may have a somewhat milky-white appearance from an excess of phosphates, mixed with more or less mucus, as in catarrh of the bladder.

When the urine is passed in large quantities, and is of a very pale straw color or has a slightly greenish tinge, it will often contain sugar, or, in other words, be the urine of diabetes mellitus, or of glycosuria from other causes. The facts that it remains markedly acid for a long time after it is passed and that it has a high specific gravity point still more to its being diabetic, and the diagnosis is confirmed if the characteristic reaction with Haines's, Whitney's, or Fehling's solution is obtained. (See Tests in this chapter.)

The following table, based on Purdy's well-known work on Urinary Analysis, sums up these conditions and their significance:

COLOR.	CAUSE OF COLORATION.	PATHOLOGICAL CONDITION.
Nearly colorless.	Dilution, or diminution of normal pigments.	Nervous conditions: hydruria, diabetes insipidus, contracted kidney.
Dark yellow to brown red.	Increase of normal, or occurrence of pathological, pigments.	Acute infectious diseases.
Milky.	Fat-globules.	Chyluria.
	Pus-corpuscles.	Suppurative diseases of the urinary tract.
Orange.	Excreted drugs.	Santonin, chrysophanic acid, senna.
Red or reddish.	Unchanged hæmoglobin.	Hæmorrhages, or hæmoglobinuria.
	Pigments in food (logwood, madder, bilberries, fuchsin).	
Brown to brown black.	Hæmatin.	Small hemorrhages.
	Methæmoglobin.	Methæmoglobinuria.
	Melanin.	Melanotic sarcoma.
	Hydrochinon and catechin.	Carbolic-acid poisoning.
Greenish yellow, greenish brown, approaching black.	Bile-pigments.	Jaundice.
Dirty green or blue.	A dark-blue scum on surface, with a blue deposit, due to an excess of indigo-forming substances. Indol. Methylene blue.	Cholera, typhus; seen especially when the urine is putrefying.
Brown yellow to red brown, becoming blood red upon adding alkalies.	Substances which are introduced into the system with senna, rhubarb, and chelidonium.	

Specific Gravity. After having examined the urine with the naked eye and by suitable tests, the physician proceeds to study it by the aid of the urinometer or specific gravity apparatus. This consists of a tall, straight glass into which the urine is poured at the temperature of 60° F. The specific gravity flask is now gently lowered into this fluid, and allowed to float in it freely, care being taken that no bubbles of air adhere to the bulb or to the upper end of the flask, since these will float it and help to raise it out of the urine, thereby giving a fictitious reading of the scale on its stem. The urine used in the urinometer should be, if possible, a sample from the total amount passed in twenty-four hours, since the specific gravity of this secretion varies at different times of the day, and a single specimen may give an incorrect idea of the real specific gravity of the total amount of the fluid. The normal specific gravity varies from 1015 to 1025.

The specific gravity of the urine varies from 1005 to 1040 at 60° Fahr.; but a persistently low specific gravity indicates chronic contracted kidney if no dietetic cause can be found, while a persistently high specific gravity either shows concentration of the urine as the result of fever or parenchymatous nephritis, or, if the urine is very light in color, the cause is perhaps diabetes mellitus, the high specific gravity being due to the sugar which it contains.

When the physician is desirous of determining the total amount of solids excreted in twenty-four hours from the specific gravity the precaution of taking the specific gravity of the total quantity of urine is particularly important. (See below.)

The Total Quantity of Solids excreted by the kidneys in twenty-four hours can be roughly estimated by taking the specific gravity of the total quantity of urine passed in twenty-four hours, and then multiplying the last two figures of this specific gravity by 2. The number obtained represents the total amount of solids in 1000 c.c. of urine. Thus supposing the urine to have a specific gravity of 1025, we would find that $25 \times 2 = 50$. Instead of this means, we can also resort to what is known as Haine's modification of Häser's method, although if the solids are much decreased, more accurate methods of testing should be resorted to. The method just referred to is carried out as follows: the last two figures of the specific gravity of the urine are multiplied by the number of ounces voided in twenty-four hours, and the product is multiplied by 1.1. Thus, if a patient passes 32 ounces, and the specific gravity of the urine is 1.012, we multiply 32 by 12, which equals

384, and this is multiplied by 1.1, which equals 422, which would be much less than the normal for a person of, say, 150 pounds, who should pass about 945 grains of solids in twenty-four hours.

This estimation of the total quantity of solids eliminated in the twenty-four hours is of great diagnostic and therapeutic value. It has been found as the result of an immense amount of research that a healthy person of about 145 pounds' weight, and from twenty to thirty years of age, taking an ordinary diet and ordinary exercise, should eliminate 61.14 grammes or 945 grains of solids in twenty-four hours. In the patient between forty and fifty years of age, we must deduct 10 per cent. from this. If between fifty and sixty, 20 per cent.; if between sixty and seventy, 30 per cent., and in patients over seventy, 50 per cent. If a patient is on a very light diet, we must deduct 15 per cent. from the average solids, and if at rest and free from exercise, 10 per cent. If it be found that a patient is eliminating much less solids than normal, we should consider that he has renal inactivity, and it is our duty to give diuretics. This is particularly the case in fevers, in which there is great tissue-waste, so that the solids ought to be greatly increased above the normal. If they are not eliminated, danger is present from their retention. (For accurate estimation of urea, see Chemical Tests.)

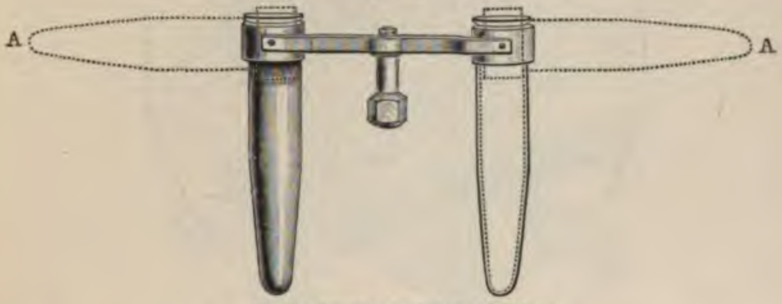
As a further guide to determine the activity of the kidney, we may employ the so-called methylene-blue test, which consists in injecting into one of the muscles of the thigh 1 grain of methylene-blue in 10 minims of water. Ordinarily the blue appears in the urine within an hour or two, and disappears within thirty-six to forty-eight hours; whereas in disease of the kidney, its primary appearance is delayed, and it remains present for several hours because the kidney is not competent to speedily eliminate it from the system. This indicates that the kidney is also inactive in regard to other eliminative functions.

MICROSCOPIC APPEARANCE OF THE URINE AND ITS CONTENTS. Having considered the macroscopical appearance of the urine, we may turn to its microscopical appearance, and this part of the subject is of even greater importance than the study of the gross appearance of this secretion, for, very commonly, a sample of urine which looks quite normal to the naked eye is loaded with microscopic objects of the greatest pathological significance. The most important of these objects are what are called "casts"—that is, moulds of the uriniferous tubules, formed as a result of the disease-process present in the kidney. These casts consist of epithelial cells, blood-

and pus-corpuscles, masses of micro-organisms, or of broken-down organic matter, as in fatty casts, and in hyaline or transparent bodies, or moulds which are made up of unknown material, but often covered by corpuscles, pus-corpuscles, or epithelial cells. In addition to these bodies we find a large number of organic bodies or derivatives of organic matter, and inorganic substances derived from the tissues or from food.

The physician who desires to examine urine successfully by the aid of the microscope must bear in mind that it can be examined satisfactorily after it has stood still in a glass or other vessel for a long enough time to allow sedimentation to take place—that is, until the objects floating in the fluid have had time to settle, or employ a more rapid method of obtaining the sediment by the use of the centrifuge, an apparatus by means of which the solids in a fluid are separated by centrifugal force. By the use of this apparatus a sediment can be obtained in a few minutes after the urine is passed. (See Fig. 173, and for a description of the apparatus see chapter on Blood.)

FIG. 173.

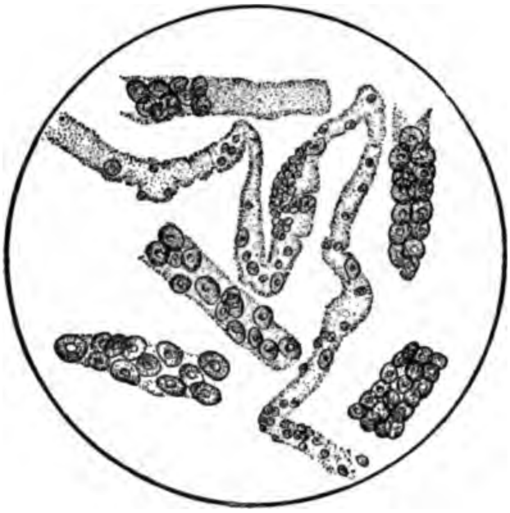


Holder for urine-tube in centrifuge.

The sediment is to be drawn up into a pipette which has been introduced into the urine and a few drops placed upon a glass slide, after which the drops are to be covered by a cover-glass and the slide placed under the microscope.

Casts composed of epithelial cells present an appearance similar to that seen in Fig. 174, and are due to proliferation or exfoliation of the epithelium lining the uriniferous tubules. The cells look swollen and granular and may contain globules of fat. These epithelial casts occur in three forms: first, they may appear as hollow casts of the tubule when the epithelium has exfoliated *en masse* (that is, the lining of the tube is cast off in one piece); second, they appear as casts made up of epithelial cells glued to one another; and, third, the cells are attached to the surface of a clear, transparent basis, looking like a hyaline cast. All these varieties are highly refractive of light and are not altered by chemical substances as easily as are the other casts about to be described.

FIG. 174.



Casts containing epithelial cells. (PEYER.)

Having found bodies of this sort in the urinary sediment, what is their significance? They are a positive sign of an inflammatory process in the parenchyma of the kidney, or, in other words, of parenchymatous nephritis.

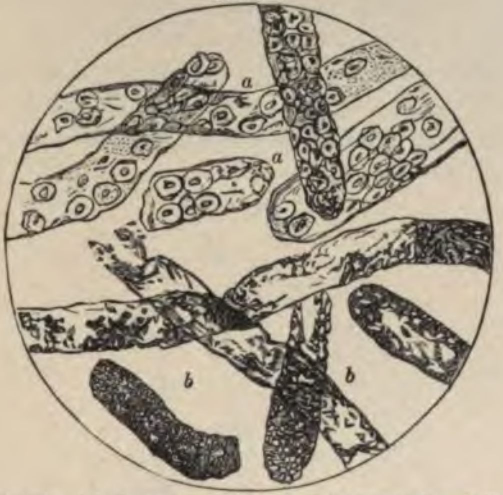
Blood-casts consist of more or less well-preserved blood-corpuscles attached to one another in a mould of the tube in which they have escaped. They are rarely seen and are masked by freely floating cells. The significance of these blood-casts is great, as they indicate an acute inflammation of the kidney, acute congestion of this organ, or a renal infarction. They are of importance, too, in separating hæmaturia arising from other sources than the kidney from hemorrhage from this organ, because they are not found unless the escape of blood has been into the uriniferous tubules.

Casts composed of pus-corpuscles are still more rarely seen, but, if constantly present, may indicate multiple abscess of the kidneys.

When masses of micrococci become grouped together in the tubules they may be expelled in casts, and under a low power look somewhat like granular casts (see below). They can be seen to consist of micrococci if a higher power is used, and they are not quickly changed by acids, as are casts composed of other materials.

The significance of their discovery is that septic infection of the kidney is present, as the result, it may be, of septic embolus brought from a distant infected part. They are seen in suppurative renal inflammation and in cases of pyelonephritis in which the true renal tissues are being involved by an extension of the disease.

FIG. 175.



a, a. Epithelial casts. *b, b.* Opaque granular casts from a case of acute Bright's disease. (ROBERTS.)

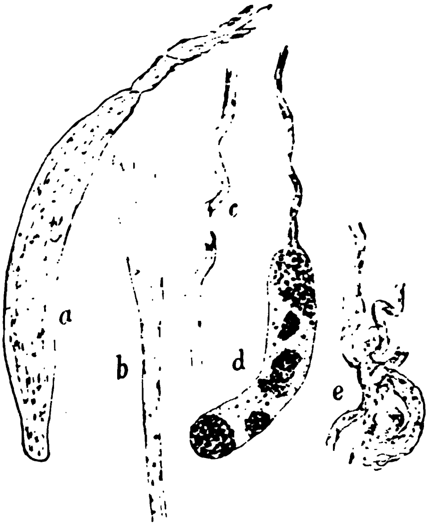
Casts, composed of broken-down organic matter, are found as granular and fatty bodies; that is, they represent broken-down blood-corpuscles and epithelial cells, and their appearance varies greatly according to the stage of the process and the origin of the materials composing them. Thus, the granular appearance may be very fine, as shown in Fig. 175, or light and refractive, dark or opaque. Very often the edges of these casts are irregular and the ends frayed and uneven. The color of these bodies may be yellow, brown, or grayish.

The significance of granular casts is not as positive as those named so far, but they often indicate a slow degenerative process in the renal parenchyma.

Fatty casts, composed of minute globules of oil, cohering to one another or attached to a central core of epithelium, or fat-crystals, are found in cases of widespread fatty degeneration, as the result of disease or poisoning, as in the case of large white kidney, on the one hand, or phosphorus, arsenical, antimonial, or iodoform-poisoning, on the other. They show the presence of a very slow process if due to disease, but have not the same significance if caused by poison. (Plate XX., Fig. 1.)

Hyaline casts are long, worm-like, transparent bodies, with very fine granulation, particularly along the edges, and because they are transparent they are often hard to find. These bodies are supposed to be composed of albumin which has been exuded into the tubules. Their significance is exceedingly grave, as they point very strongly to that incurable malady, chronic interstitial nephritis. If these casts are very large, they may show amyloid degeneration of the kidney. They have often been wrongly called "waxy" casts. (Plate XX., Fig. 1.)

FIG. 176.

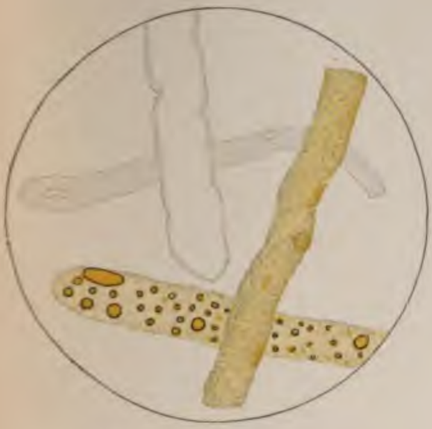


Cylindroids from albuminous urine. *a*, *b*, and *c*. Ribbon-like forms. *d*. Cast-like form, with cells upon its surface. *e*. Filamentous forms in a clump.

Casts are not to be confused with cylindroids or streamers. These cylindroids appear in several forms. Most commonly they look like threads or filaments which are transparent and often somewhat striated or hyaline in appearance. They are often long enough to extend

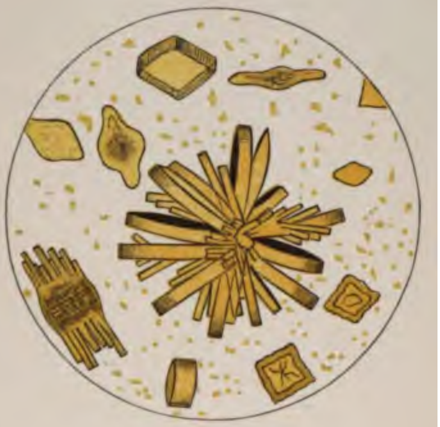
PLATE XX.

FIG 1.



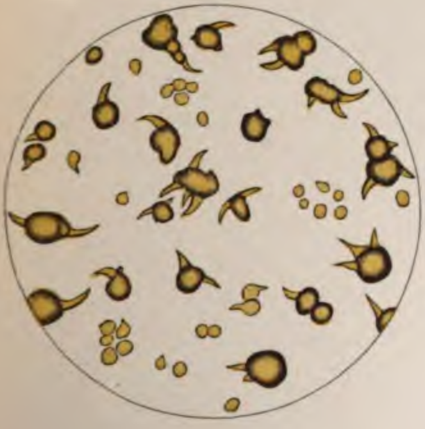
Casts, Fatty, Waxy, Hyaline and Granular.

FIG. 2.



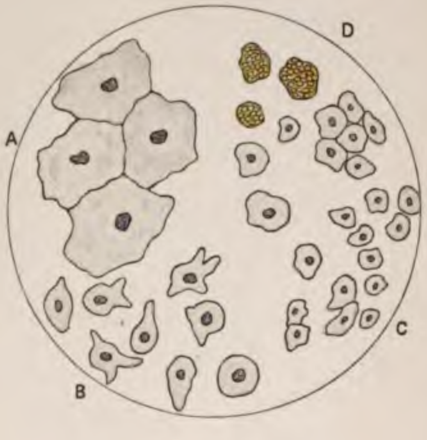
Crystals of Uric Acid.

FIG. 3.



Ammonium Urate Crystals.

FIG. 4.

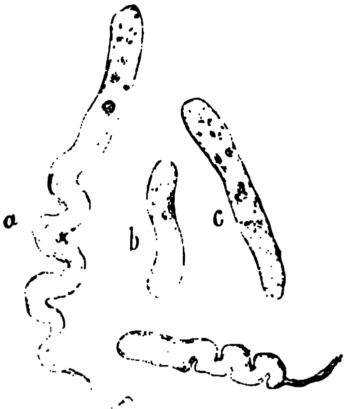


Epithelial Cells.

A, squamous epithelium; B, bladder epithelium; C, kidney epithelium; D, kidney epithelium (fatty).

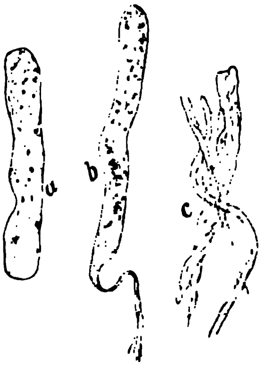
completely across the microscopic field, and if followed out to the end will be found to taper off or gradually become more and more transparent until they cannot be outlined. For this reason too much light should not be used in searching for them, nor should a lens of too high a power be used. These cylindroids often are grouped in bunches. In other instances we find cylindroids in the form of

FIG. 177.



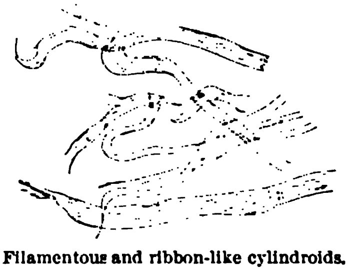
Non-albuminous urine.
Cast like forms with deposit of urates.

FIG. 178.



Non-albuminous urine.
a and *b*. Cast-like forms. *c*. Filamentous.

FIG. 179.



Filamentous and ribbon-like cylindroids.

ribbons, or, in other words, they are wider than the thread-like masses just described. In still other instances the resemblances to true tube-casts are so marked that a differentiation is scarcely possible, except that they are sometimes found to have a filiform tail-like ending. (See Figs. 176, 177, 178, and 179.) The significance of cylindroids is not definitely known, but they may be taken as an indication of irritation of the kidneys, even if albumin and true casts cannot be found in the urine. They are often seen in the renal irritation following or, rather, accompanying the conditions called lithæmia or uricæmia, and in that condition in which we find oxaluria. (For further information concerning cylindroids, see Stengel's paper in *The Medical News* of July 15, 1893.)

According to Bramwell, the following is the best method of staining and mounting tube-casts and other urinary deposits. He uses picrocarmine.

" 1. An ordinary conical urine-glass is filled with equal parts of urine and an aqueous solution of boric acid, and set aside until the deposit settles.

" 2. The deposit is then drawn off by means of a pipette, and transferred to an ordinary test-tube, in which a small quantity (half a drachm is quite sufficient) of picrocarmine solution has been previously placed.

" 3. The urine and staining-fluids are then thoroughly mixed by inverting the test-tube two or three times, the end being closed, of course, by the thumb.

" 4. The test-tube containing the urine and staining-fluid is then set aside to stand for twenty-four hours.

" 5. The deposit, which has by that time settled at the bottom of the test-tube, is then drawn off by a fine-mouthed pipette, placed on a slide, covered, and examined under a low power.

" If any tube-casts are present, they are very easily detected by this method.

" When a cast is detected, it should be carefully brought to the centre of the field and examined with a higher power. If amyloid degeneration is suspected, methyl-violet may be used, for in some cases of waxy disease of the kidney the tube-casts give the characteristic rose-pink reaction with methyl-violet. For permanent preparation the deposit is drawn off as in No. 5, above, and transferred to a small tube of Farrant's medium,[1] in which it remains until the organic deposit has settled, when it is again drawn off and transferred to clear Farrant's solution, whence it is mounted in the usual manner. All organic deposits are thus stained and mounted in a perfectly clear medium. Their minute characters can be studied with the highest powers of the microscope."

---

[1] Farrant's solution is made as follows: Dissolve 1 grm. of arsenous acid in 200 c.c. of distilled water. In this dissolve 130 grms. of gum acacia with frequent stirring, and add 100 c c. of glycerin. Filter the solution through fine Swedish paper upon which has been deposited a thin layer of talc.

The most important sedimentary substances for diagnostic purposes, other than casts, are the products of tissue-changes, or are derived from articles of food. These substances are chiefly the acid urate of sodium and potassium and alkaline urate of ammonium and potassium, uric acid, oxalate of lime, the phosphate, carbonate, and sulphate of lime, and the so-called triple phosphate (ammonio-magnesic phosphate).

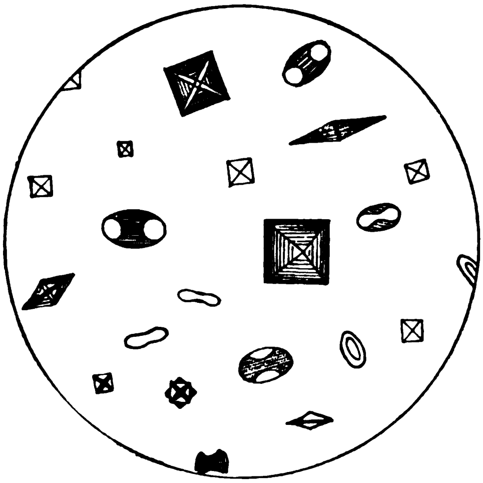
The discovery in a urinary sediment of fine shapeless granules, which may be crystalline and shaped like a fan, which are generally brown or pinkish in hue, indicates acid sodium urate. Urine containing such deposits is found to become acid on standing, and will form a brick-dust deposit as soon as it is cooled. Acid potassium urate and acid calcium urate, which occur in an amorphous form, are mixed with it in smaller quantities.

The urates themselves have no particular importance except that they are often present in excess in fever, wasting diseases, gastric disorders, and in attacks of gout.

When in a highly acid urine the student finds rhombic or diamond-shaped plates (Plate XX., Fig. 2), or plates of a similar shape with the lateral angles rounded off, or quadrate crystals or square plates, or plates like double-headed arrows, or rosettes of crystals, or bundles of crystals like bundles of kindling-wood, these forms are uric acid. Any urine will deposit such crystals if it stands for many hours (say ten hours), as its acidity increases, and therefore the discovery of these crystals only possesses significance if they are found in from four to six hours, as this shows an excess of uric acid, which in turn is found in gouty or rheumatic persons or in those who eat to excess and take no exercise. Often an excess of uric acid in the urine antedates the development of chronic contracted kidney. Uric acid also appears in excess in cases suffering from fever and acute inflammations. It is also eliminated in excess in leukæmia, splenic enlargement, hepatic cirrhosis, and gastro-intestinal catarrh. The rosette crystals just named are often found in diabetic urine.

Small, square, brilliant octahedral crystals which are perfectly transparent and refract light strongly, looking somewhat like the back of a square envelope at times, are those of oxalate of lime. (See Fig. 180.) The significance of oxaluria is quite important, for it is often a concomitant symptom of melancholia depending upon defective metabolism. The finding of oxaluria separates this class of cases from those of the true disease melancholia, and indicates the use of nitrohydrochloric acid. These crystals are, however, found in the urine of persons who have eaten pears, cabbage, or tomatoes, and in that of persons suffering from spermatorrhœa. If not due to the ingestion of the foods named, oxaluria indicates deficient oxidation of nitrogenous tissues.

FIG. 180.



Oxalate of lime crystals.

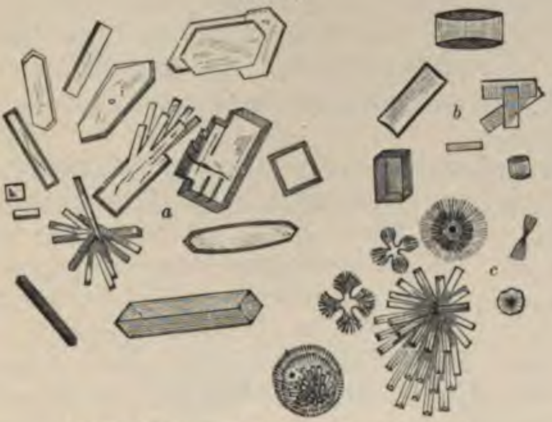
Creatin in the urine occurs in very brilliant prisms of a rhomboid form, the end of which is often split into a frayed end. (See Fig. 181, *a*.) It is not present in normal urine.

Creatinin also exists in normal urine in small amounts in prismatic, colorless, brilliant crystals of the shape shown in Fig. 181, *b*.

When dark-brown spherical masses covered with thorn-like crystals or sharp spicules are formed in alkaline urine, they are composed of ammonium urate (Plate XX., Fig. 3), and they will be found associated with crystals which are flat or shaped like coffin-lids, or more rarely are feathery, star-shaped masses which are large in size. These are the crystals of the triple phosphate. (See Fig. 182.) In addition, such urine will contain amorphous calcic phosphate.

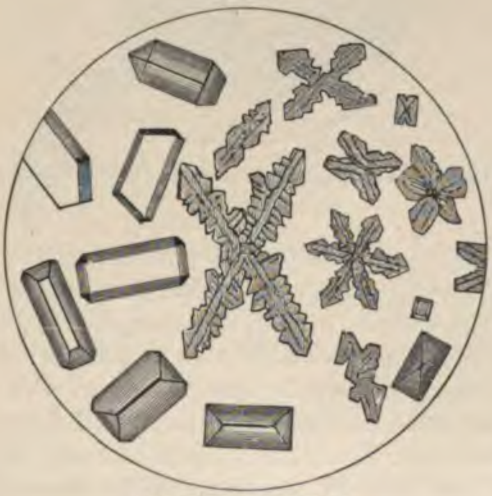
The crystals of the triple phosphate are of some diagnostic importance, as they do not exist in the normal urine, but are formed when ammonia is set free by the decomposition of the urea. If such crystals are found in freshly passed urine, they indicate that ammoniacal fermentation is taking place in the bladder, a condition often seen in chronic cystitis and in some cases of paraplegia arising from injury to the cord or myelitis. A deposit of the triple phosphate and amorphous calcium phosphate, making a sediment like that of purulent urine, is sometimes seen in persons suffering from overwork of the nervous system and in cases of general debility.

FIG. 181.



Crystals of creatin and creatinin. (CHARLES.)
a. Crystals of creatin. *b.* Crystals of creatinin. *c.* Crystals of chloride of zinc and creatinin.

FIG. 182.

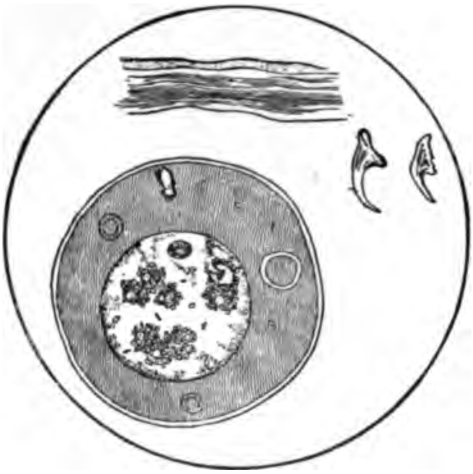


Triple phosphate crystals.

In addition to these amorphous and crystalline bodies found in the urine there are a number of others derived from the body, or due to extraneous contamination. These are epithelial cells derived from the kidneys, ureters, bladder, or urethra (Plate XX., Fig. 4). Eggs or bodies of several parasites, tubercle bacilli, gonococci and streptococci, or staphylococci are also sometimes seen under the microscope. In addition, we find spermatozoa in certain cases. (See Fig. 185.)

Thus, we may find the embryos of filaria, echinococcus hooklets (Fig. 183), and the eggs of distoma hæmatobium, which are very rarely seen.

FIG. 183.

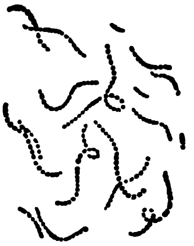


Echinococcus, with two hooklets, and section of cystic membrane, greatly magnified. (PEYER.)

Tubercle bacilli are to be found by the same staining-processes as when they are sought for in the sputum (see chapter on Cough and Expectoration), and, if found in the urine, indicate renal or vesical tuberculous infection, provided that the patient has not contaminated the vessel holding the urine by sputum infected with the organism. They are not to be confused with the bacilli found in preputial smegma, which look like tubercle bacilli and take the same stains.

Gonococci indicate the presence of a specific urethritis or vaginitis, and are found by staining and using a $\frac{1}{12}$ homogeneous immersion lens with a No. 2 eye-piece. The process of staining is by the use of eosin and methylene-blue. The material on the cover-glass is stained for a few seconds in an alcoholic solution of eosin, then the excess of stain is washed off, and the slide is placed for ten minutes in an aqueous solution of methylene-blue. Streptococci appear in chains and are stained by the same process. They show infection from pus and are found in cases of erysipelas. (See Fig. 184.) Staphylococci also indicate pus-formation in the urinary tract.

FIG. 184.

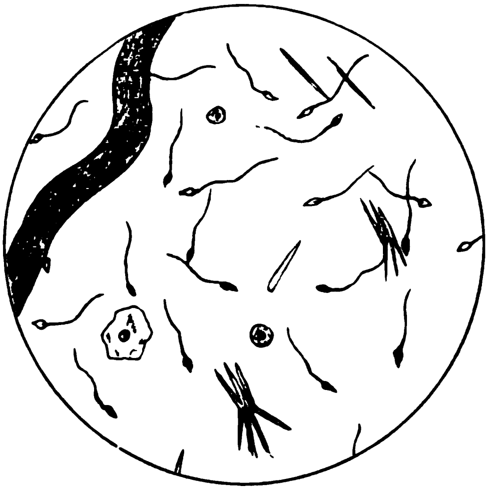


Streptococci. (ABBOTT.)

The presence of spermatozoa is not so common as is generally thought. They may be in the urine either as the result of a true spermatorrhœa, which is rare, or from some of the semen remaining in the urethra after an ejaculation in coitus, or from an emission at night without intercourse. They appear as small, transparent bodies having a head and tail, and, if alive, possess very active movements. (Fig. 185.)

Fermentation resulting from the presence of a number of special fungi takes place in both healthy and diseased urines after they are

FIG. 185.



Spermatozoa, with casts of seminal tubules and spermine crystals.

passed. In normal urines the acidity, which is generally present to a slight degree, becomes still more acid through the growth of a special fungus. This process is accompanied by the deposition of uric acid, acid sodium urate, and calcium oxalate, and also amorphous urates. After the urine is exposed still longer it undergoes an alkaline fermentation, and there develop in the fluid the micrococcus ureæ and bacterium ureæ. As a result, the urea takes up water and decomposes with the development of CO_2 and ammonia. No sooner is a positive alkaline reaction established than those ingredients of the urine which are insoluble in an alkaline solution are precipitated, namely, amorphous calcic phosphate, ammonium urate, and ammonio-magnesic phosphate. The first is amorphous, but the ammonium urate appears under the microscope in the form of small granules of a dark color which are covered with spines. The crystals of ammonio-magnesic phosphate are shaped like a coffin-lid and are large.

The third form of fermentation taking place in the urine is that which occurs in diabetic urine, and is due to saccharomyces albicans, the micro-organism which produces fermentation in ordinary solutions of glucose.

Chemical Tests. The chemical tests of the urine give us much important information. We commonly test it for albumin and for sugar, and if we wish still further information, we examine it for its percentage of urea, uric acid, and for its peptones, or, to use a better term, its albumoses.

Urine to be subjected to chemical examination should always be filtered if strictly accurate results are sought.

ALBUMINURIA. There are a great number of tests for albumin and sugar. Many of them are open to fallacies, and they are, therefore, to be avoided by the busy practitioner, who can rest assured that if he finds no albumin by the heat and nitric acid test, if properly carried out, he can put albuminuria of pathological significance out of the possibilities of the case, provided he tests samples taken at different times and on several different days, for sometimes albuminuria is intermittent.

The boiling test consists in taking filtered urine and pouring enough of it into a perfectly clean test-tube to fill it about two-thirds. If the urine be turbid by reason of an excess of urates, the fluid can be rendered clear by gently heating all of it. To this urine are now added a few drops of acetic acid to render it acid; for if neutral, the albumin will not be coagulated by heat. The upper part of this urine is now boiled by holding it over an alcohol lamp, and if albumin is present a fine cloud will appear in the boiled part of the urine, while the lower part remains clear. This cloud may be due to albumin or to earthy phosphates. If a drop or two of nitric acid is allowed to trickle down the side of the tube, the cloud is dissipated if due to phosphates, but not changed if due to albumin.

If acetic acid is employed in the place of nitric acid for the purpose of acidifying the urine, care must be taken that it is not used in excess, as under these circumstances the albumin will be dissolved. Indeed, this possibility of the solution of acid albumin is one of the fallacies in the boiling test which may be avoided if, after acidifying the urine until it is distinctly acid, one-sixth of the urinary volume of a saturated solution of common salt, sodium sulphate, or magnesium sulphate, is added to it, when, upon boiling, the albumin is precipitated. This test, performed with these modifications, discovers any albumin which may be present, even if its amount be very small. If upon the addition of the saturated solution of common salt a precipitate is formed, this may be due to the presence of albumoses, but the albumose precipitated is redissolved on boiling.

Another useful, rapid, and moderately accurate test for albumin is the so-called nitric acid test. Two or three drachms of urine are placed in a test-tube, and then a small quantity of pure nitric acid is allowed to trickle down the side of the tube, in which, by reason of its greater specific gravity, the acid passes to the bottom, the urine forming a supernatant layer. If serum-albumin is present, a distinct white cloud will appear in the form of a ring at the point of contact between the two liquids. A much more effective way of performing this test is to use a conical glass, somewhat like a sherry glass, placing in it 20 cubic centimetres of urine, and then by means of a pipette, which is carried to the bottom of the vessel, allow half that quantity of nitric acid to escape. This modification of Heller's test makes a line of albumin quite readily seen. Where only a small quantity is present, it may be necessary to allow the two liquids to remain in contact for several minutes. If an excess of uric acid be present, five or ten minutes after the addition of the nitric acid a sharply defined ring, somewhat similar to that due to albumin, will be found a short distance above the line of contact. Another way is to place the acid in the tube first and allow the urine to flow on it.

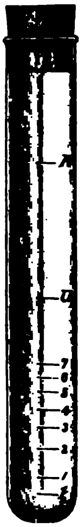
When nitric acid is added to the urine after it has been boiled, the urine sometimes becomes distinctly yellow, and, upon cooling, a white precipitate appears. Under these circumstances the presence of albumose is indicated. This precipitate is to be separated from the dark-brown coloration sometimes due to uric acid, which develops after a considerable length of time in urine which has been treated in the manner described. It is to be remembered that albumoses are precipitated by heat, but redissolved when the temperature approaches the boiling-point.

The potassium ferrocyanide test consists in strongly acidifying a few cubic centimetres of urine with acetic acid, and then adding a few drops of a 10 per cent. solution of potassium ferrocyanide. If albumin is present in quantity, a flaky precipitate occurs. If present in minute quantity, the solution becomes turbid. If the urine is very concentrated, it should be diluted with water before this test is employed. In this test, also, the precipitate may be due to albumose, and it will disappear upon boiling. If the precipitate only partially disappears, it is fair to assume that both albumose and albumin are present. The development of a cloud in the urine after the acetic acid is added and before the potassium ferrocyanide is used, does not indicate either albumose or albumin.

The trichlor-acetic acid test is more delicate than any of those which have been described. Indeed, it is so delicate that it frequently discovers albumin in urine which is certainly normal. By means of a pipette, one to two cubic centimetres of a watery solution of trichlor-acetic acid, specific gravity 1.147, are conveyed to the bottom of a test-tube holding several cubic centimetres of urine which has been carefully filtered. If albumin is present, a white ring will be seen to form at the line of contact between the trichlor-acetic acid below and the urine above. This line may be due to albumose, but is again to be differentiated by the fact that it disappears on boiling if due to this substance. Sometimes uric acid produces a somewhat similar line to that caused by albumin, but it also disappears on heating, and its appearance can be prevented by using a urine which is fairly well diluted.

The quantitative tests for albumin are many of them impractical for the busy doctor. The best method is by means of percentage-tubes placed in a centrifuge-machine. By this means all the albumin is thrown down. The tubes are filled to the 10 c.c. mark with urine and $2\frac{1}{2}$ c.c. of potassium ferrocyanide solution (one part to ten) are added. Next we add $1\frac{1}{2}$ c.c. acetic acid and thoroughly mix all these liquids, and the tube being placed in the centrifuge the machine is worked till all the albumin has settled. Each $\frac{1}{10}$ c.c. mark on the tube represents 1 per cent. by bulk of albumin; that is, if the albumin extends up to the 3½ cubic centimetre mark the albumin amounts to 35 per cent.

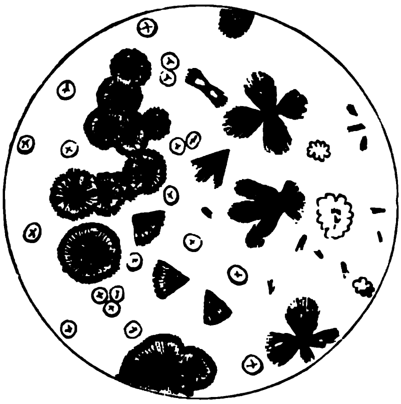
FIG. 186.



Albuminometer.

Another useful method is that known as Esbach's, in which his albuminometer is employed. A special test-tube, known as an albuminometer, is used, on which is marked a letter "U" and a letter "R." (Fig. 186.) The urine, which has been carefully filtered, is poured in the tube until it reaches the letter U, this portion of the tube being also provided with a scale reading from 1 to 7. The reagent employed is then poured in until the letter R is reached, when the tube is closed with a stopper, and it is turned up and down a number of times, so that the fluids may become thoroughly mixed. It is then set aside for twenty-four hours, when the serum-albumin, serum-globulin, albumose, uric acid, and kreatinin, will have been precipitated. The amount of these solids in grammes per thousand may be then directly read off from the scale. The reaction of the urine to be tested should be acid, and, if necessary, acetic acid must be added to it for the purpose of acidifying it. Further, its specific gravity should not be over 1.006 or 1.008. If the specific gravity is higher than this, it must be reduced by the addition of water. The temperature should be in the neighborhood of 70° F. Esbach's

FIG. 187.



Crystals of kreatinin-zinc chloride. (SALKOWSKI.)

reagent is composed of 10 grammes of picric acid and 20 grammes of citric acid dissolved in a litre of distilled water.

The significance of albumin is not as grave in all cases as it was considered at one time, nor is its quantity of great import necessarily, for in some of the gravest cases of renal disease, as chronic contracted kidney, it is excreted in very small amount, and it occurs in the urine sometimes in large quantities without any kidney lesion being present. As a rule, however, it indicates renal disease in one of its inflammatory forms, provided it is associated with other renal symptoms. It may depend on changes in the blood in which the diffusibility of its albumin is increased (Semmola), and we see albuminuria in cases of anæmia and in convalescence from protracted illness or from the effects of poisons. Again, circulatory changes may cause albuminuria by causing congestion of the kidney, as in cases of failing heart from its various causes. There is an intermittent, little-understood form of albuminuria, called cyclic albuminuria, or the albuminuria of adolescence, in which the albumin is absent on rising from bed in the morning, but appears if exercise is taken. An excess of albumin in the diet may cause albuminuria, which is not necessarily indicative of renal disease. Traces of albumin may be due to the presence of a little pus arising from cystitis or urethritis, or from a vaginitis. In the presence of albuminuria the real test to determine the presence of actual kidney disease rests upon the discovery of casts on microscopic examination. Even the absence of casts does not negative the possibility of nephritis, for they may be absent, yet renal disease be present.

ALBUMOSE IN THE URINE. Albumoses in the urine may be discovered by the use of certain tests, but even if they are found their presence has no definite significance.

Harris asserts that albumosuria is simply a manifestation of the action of micro-organisms, and is thus only an indication of an infective process: but Fitz holds that its persistent presence nearly always points to a fatal ending. It ought not to be forgotten that albumosuria occurs in the normal puerperium.

Under the name of myelopathic albumosuria, Bradshawe has described a very rare condition in which, with thinning of the bones and overgrowth of gelatinous masses in the cancellous tissues, there develops an extraordinary degree of albumosuria. The patient becomes exceedingly weak and feeble, and finally dies from exhaustion or intercurrent maladies. The peculiarity of albumosuria is that the albumose coagulates below the boiling-point and dissolves on boiling. Mineral acids precipitate it in the cold, but boiling dissolves the precipitate.

Peptonuria, at one time considered pathologically identical with albumosuria has been separated from the latter state by Chittenden's researches. It is discovered by saturating the urine, which has been first slightly acidified with acetic acid whilst boiling, with ammonium sulphate, and filtering out any precipitate. If the filtrate contains a substance which is precipitated by potassio-mercuric iodide or picric acid, it is peptone. Peptone is not precipitated by saturation with ammonium sulphate, and albumose is precipitated by it. Peptonuria is present in croupous pneumonia, all suppurative processes, empyema, tuberculosis, smallpox, mumps, erysipelas, cancer of the viscera, jaundice, and apoplexy, and in typhoid fever and phosphorus-poisoning. Von Jaksch asserts that it is present in epidemic cerebrospinal meningitis and absent in tubercular meningitis, and that it is a positive differential sign of the former disease if no ulceration of the lungs is present. In the presence of acute inflammations the development of peptonuria indicates suppuration.

SUGAR IN THE URINE. The presence of sugar is determined by a large number of qualitative and quantitative tests, of which the simplest and most reliable are Haines's test and the test of Whitney. Haines's test consists in making a solution as follows: pure copper sulphate, thirty grains; distilled water, half an ounce; thoroughly dissolve the copper salt in the water; add pure glycerin, one-half ounce, which is to be thoroughly mixed; and then add liquor potassæ, five ounces. One drachm of this is to be placed in a test-tube and gently boiled, and to this are added six to eight drops of the urine, and the liquid again gently boiled. If sugar is present, a copious yellow precipitate is formed. This is better than Fehling's test, because it is a permanent fluid.

Whitney's test is a solution of ammonio-cupric sulphate, of which one drachm is decolorized by $\frac{1}{30}$ grain of glucose. The solution of the amount of one drachm is placed in a test-tube and heated to the boiling-point. The urine is now added drop by drop. If no sugar is present, no change will occur; but if it is, the blue color will begin to fade, and finally the liquid will become perfectly colorless. As the fading process begins the urine should be added more slowly, three to five seconds of boiling intervening between each drop. If there is any shade of blue or green left in the solution, reduction has not taken place. The following table shows how this test may be used for the quantitative estimation of sugar:

If reduced by	*It contains in the ounce.*	*Percentage.*
1 drop	16 or more grains.	3.33
2 drops	8	1.67
3 "	5.33	1.11
4 "	4	0.83
5 "	3.20	0.67
6 "	2.67	0.56
7 "	2.29	0.48
8 "	2	0.32
9 "	1.78	0.37
10 "	1.60	0.33

If the urine contains more than 3.33 per cent. of sugar, it is to be diluted by from one to ten parts of pure water, and the amount found in the table multiplied by the amount of dilution.[1] Usually diabetic urine contains not less than half of 1 per cent. and rarely more than 1 per cent. This test is not reliable if the solution is not freshly prepared.

As Fehling's test is so widely used it must be mentioned. Wickham Legge thus describes it:

This solution may be prepared in the following way: 665½ grains of crystallized potassio-tartrate of sodium are dissolved in five fluidounces of a solution of caustic potash, sp. gr. 1.120. Into this alkaline solution is poured a fluid prepared by dissolving 133⅓ grains of sulphate of copper in ten fluidrachms of water. The solution is exceedingly apt to decompose, and must always be kept in stoppered bottles and in a cool place. It is usually, therefore, more convenient not to mix the alkali and copper until the solution is wanted for use. In this case a fluidrachm of the sulphate of copper solution may be added to half a fluidounce of the alkaline solution prepared as above.

About a couple of drachms of this test-solution are poured into an ordinary test-tube, and the fluid boiled over a lamp and set aside for twelve hours. If no deposit forms, the solution may be used for analysis; but if a red precipitate be thrown down, the liquid has decomposed, and a fresh supply must be had.

While the solution is boiling in the test-tube the urine must be added to it drop by drop, and the effect watched. A few drops of a sample of urine which contains a large percentage of sugar will at once give a precipitate of yellow or red suboxide of copper; but if no precipitate occur, the urine should be added to the fluid drop by drop, any deposit being carefully looked for, until a quantity equal to that of the Fehling's solution employed has been added. If no precipitate be found after setting the test-tube aside for an hour, the urine may be considered free from sugar.

Cautions: 1. The test-solution should never be used without boiling beforehand for a few seconds, the tartrate being exceedingly apt to decompose, and the solution then reduces copper as effectually as would grape-sugar.

2. The quantity of urine used in the test should never be greater than the quantity of test-solution employed.

3. After adding urine in volume equal to the Fehling's solution, the boiling of the mixture must not be continued, as other bodies present in the urine, beside sugar, will reduce copper at a high temperature.

If the examination for sugar is to be made with the greatest care, the urine should always be filtered, at least three times, through animal charcoal. This removes all urates and uric acid, which often partly reduce the Fehling's solution, but the sugar goes through the filter.

Sir William Roberts directs that the Fehling's solution be placed in a test-tube to the depth of about one-quarter inch and the filtered urine added to the depth of two inches, and the two fluids well mixed. The flame of the lamp is then applied to the upper part of the urine, as in testing for albumin, and this is briskly boiled for a few seconds. The test-tube is now held up to the light, and, if sugar is present, the upper part has a yellowish tinge, while the earthy phosphates are thrown down in golden-colored flocculi.

Trommer's test for sugar consists in strongly alkalinizing a few cubic centimetres of urine with a solution of sodium hydrate, and then adding drop by drop a 5 per cent. solution of sulphate of copper until the cupric oxide formed is no longer dissolved. The mixture is then carefully heated, and if sugar is present, a yellow precipitate of cuprous oxide is formed which gradually settles to the bottom of the test-tube as a red sediment.

The phenylhydrazin test consists in adding to 6 or 8 cubic centimetres of urine two small pinches of phenylhydrazin hydrochlorate equalling about 5 to 7 grains, and 15 grains of acetate of sodium. This mixture is then warmed until the salts have been dissolved, a little water being added, if necessary, to aid their solution. The test-tube is then placed in boiling water for twenty to thirty minutes, and then transferred to a vessel containing cold water, when, if sugar is present in considerable quantity, a bright-yellow crystalline deposit will at once be thrown down and partly adhere to the tube. These crystals are composed of phenylglucosazon and are insoluble in water. This is less open to fallacy than any other test for sugar.

The *quantitative estimation of sugar* is best made by the Whitney test, already described, or by the fermentation-method of Roberts, which depends upon the principle that grape-sugar is decomposed into alcohol, carbon dioxide, etc., by the fermentation set up by yeast. As a result of this the urine loses its specific gravity, and each degree of specific gravity has been found to equal one grain of sugar in the fluidounce. In other words, if the specific gravity before the test was 1.035 and after the test 1.015, the amount of sugar present would be twenty grains per ounce. Four ounces of urine are placed in a twelve-ounce bottle and a lump of German yeast added. The bottle is then corked with a perforated cork to permit the gas to escape, and placed in a warm place for twenty-four hours. By its side is placed a tightly corked bottle of the same size, holding four ounces of urine and no yeast. The specific gravity of both specimens is taken simultaneously, and the difference in degrees represents the number of grains of sugar in each ounce. The loss in degrees of specific gravity multiplied by 0.23 will give the percentage of sugar.

The *significance of sugar* in the urine is various. If it is persistent and accompanied by wasting, polydipsia, and polyphagia, it is a sign of diabetes mellitus, due to a lesion in the medulla, to morbid functional activity of the liver, or to changes in the pancreas. If diabetes mellitus occurs in a young person, the prognosis as to life is nearly always fatal; if in middle age, it is hopeful; if in persons after fifty, it is quite favorable.

Sugar is sometimes found in small amounts in the urine of very obese persons, and its presence under these circumstances does not necessarily indicate a grave prognosis; but, on the other hand, there are cases of so-called diabetogenous obesity in which the prognosis is very grave. They are to be separated from the class first named by the fact that the systemic symptoms of wasting, depraved nutrition, itching, furunculosis, and profuse diuresis are present. Then, too, in the latter form, the disease is usually associated with obesity in early life, whereas in the milder form it occurs in the obesity of advanced life. Diabetes occurring in old age, or after sixty years of age, has not the grave prognosis attached to it that exists in connection with the disease in earlier life, as just stated. The younger the patient the graver the malady. (See also end of this chapter.)

The other indications of glycosuria, aside from diabetes mellitus, are of little importance. Glycosuria occurs in the course of convalescence from many infectious diseases, particularly typhoid fever, measles, scarlet fever, diphtheria, influenza, and malarial disease after cerebral hemorrhage and nervous injuries, and after the ingestion of some poisons, notably phloridzin, chloral, arsenic, alcohol, and curare. It also sometimes occurs as a result of the ingestion of large amounts of sugar and starchy foods in persons who are unable to digest and assimilate carbohydrate foods in excess. Unless the glycosuria is associated with the other symptoms of diabetes mellitus, it is not a positive sign of the disease, for glycosuria is a symptom of a number of states other than diabetes mellitus, as has just been pointed out.

The presence of an excess of *acetone* in the urine in association with glycosuria is diagnostic of true diabetes mellitus, and if the acetone is present in large quantity, the condition may be considered as grave. The best test for acetone, in that it is the simplest, is Oppenheimer's modification of Dennigés' test. It is carried out as follows: The reagent named below is added drop by drop to about 3 cubic centimetres of urine until the precipitate which is formed no longer disappears on stirring. A few more drops are now added, and after several minutes the precipitate is filtered off. The clear filtrate now has added to it 2 cubic centimetres of the reagent and 4 cubic centimetres of a 30 per cent. solution of sulphuric acid, and is boiled for a minute or placed in a water-bath of boiling water. If acetone is present in large quantity, a heavy white precipitate forms at once; while if it is present in minute quantity, a slight cloud develops on standing after several minutes. The precipitate is almost completely dissolved by adding an excess of hydrochloric acid. It is claimed that this test will reveal acetone in the presence of 1 : 5000. If albumin is present in the urine, large amounts of the reagent must be added. The reagent consists of 20 grammes of concentrated H_2So_4 added to 100 cubic centimetres of dissolved water, to which are also added 5 grammes of freshly prepared yellow oxide of mercury.

Finally, it is not to be forgotten that a condition known as *alkaptonuria* may exist. In this state the urine reduces alkaline solutions of copper, and on exposure to the air absorbs oxygen in the presence of an alkali and becomes of a dark-brown or black hue. The specific gravity of the urine is low, 1.014 to 1.020, and there is no marked polyuria. While such a urine reduces Fehling's test, it will not give the reaction with the bismuth, the phenylhydrazine, the fermentation, or polariscope tests for sugar. This condition of alkaptonuria has no direct pathological significance so far as is known. It is often found in several members of the same family.

An excess of *indican* in the urine possesses considerable clinical significance. It nearly always indicates putrefactive processes going on in the intestine, probably resulting from the putrefaction of albuminous foods. A certain amount of indican is always present in the urine, particularly if large quantities of animal food have been taken, and in the presence of obstruction it often appears in the urine in very large quantities, so that its presence in excess has been considered as a valuable aid to the diagnosis of this condition, since in ordinary constipation it does not appear in great excess. The test for indican is performed by means of Jaffé's test, as modified by Stokvis, as follows: Equal parts of urine and concentrated hydrochloric acid are placed in a test-tube, and to this mixture are added two or three drops of a strong solution of sodium hypochlorite. To this are also added one to two cubic centimetres of chloroform, and the mixture is then thoroughly shaken. The indigo which is set free is taken up by the chloroform, which becomes blue to a greater or less extent according to the quantity of indican which is present. The fallacies connected with this test are: First, that bile-pigment interferes with it. If bile-pigments are present in considerable quantity, they must be removed by the previous addition to the urine of a solution of subacetate of lead in moderate quantity. The presence of iodine, due to the use of potassium iodide, will also color the chloroform red instead of blue.

The best test for *bile* in the urine is Gmelin's test, as modified by Rosenbach. After the urine has been filtered through thick filter-paper, the filter-paper, still wet, is removed from the funnel, and a drop of concentrated nitric acid is allowed to touch its inner surface, when, if bilirubin is present, a play of colors will take place at the line of contact, the green color being most typical.

THE UREA IN THE URINE. The amount of urea is to be estimated by the process of Lyons, as follows (Fig. 188):[1]

1. A bottle is provided with a perforated rubber cork and delivery tube; in this the decomposition of the urea is effected.

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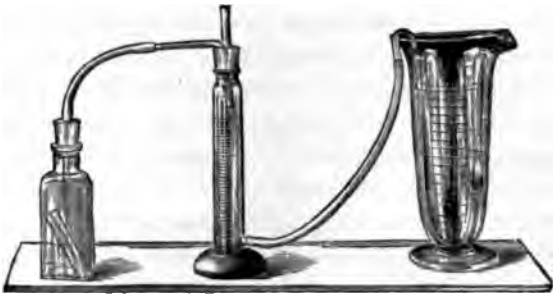
[1] This apparatus, with full directions for use, can be obtained from Parke, Davis & Company, Detroit, Mich., for one dollar.

FIG. 188.



Ureometer.

2. A small test-tube to contain the urine, graduated to hold 4 c.c., the quantity employed in each experiment.

3. A graduated jar for measuring the gas evolved. The jar is provided at the bottom with an "overflow" tube, and at the top with a vent-tube closed with a rubber cap, to secure accurate adjustment of the level of the fluid in the jar at the commencement of the experiment.

The process is as follows: put into the square bottle 20 c.c. of a special solution of chlorinated soda (for formula see below), and add 5 c.c. of a 20 per cent. solution of potassium bromide; fill the test-tube exactly to the mark (4 c.c.) with the urine to be examined, and lower it into the bottle by means of a thread or by the aid of a pair of dressing-forceps, taking care that none of its contents is spilled in the operation. Fill the graduated jar with water, which must be of the same temperature as the air of the room, to a point a little above the 0° of the scale, supporting the extremity of the overflow-tube so that no water can escape. Remove the rubber cap from the vent-tube and connect the apparatus, pressing in the rubber corks firmly so as to make the joints air-tight. Finally, put on the rubber cap, drawing it down so as to force a little water out of the overflow tube, and bring the level of the water remaining exactly to the 0° mark, the orifice of the overflow-tube being on the same level. A little practice will make this easy.

To make sure that the connections are all perfectly air-tight, lower the end of the overflow-tube a few inches; a few drops of water will escape from diminished pressure, but if the joints are perfect there will be no further dropping. If there is any leakage, the defective joint must be found and the difficulty corrected before proceeding further with the experiment. Having made sure that the connections are perfect, catch the curved end of the overflow-tube over the edge of a measuring graduate, as shown in the illustration (an ordinary bottle or any other receiver may be used in place of the graduate). Now, by canting the bottle, cause the urine to flow out of the test-tube and mix with the test-solution. Effervescence is at once produced, and the gas evolved forces a corresponding volume of water out of the overflow-tube. Shake the bottle occasionally to promote the escape of the gas. When the action appears to be at an end pour into the measuring graduate enough water to reach above the opening of the overflow-tube, in order that cooling of the gas evolved, which at first is quite warm, may not draw air into the apparatus. Let the apparatus stand fifteen or twenty minutes to cool, then shake the bottle containing the urine once more and proceed to read off the result. To do this, it is necessary to bring the opening at the end of the overflow-tube just to the same level as that of the fluid remaining in the graduated cylinder, since raising or lowering the tube slightly affects the volume of the gas to be measured. The percentage of urea is read off without need of any calculation from the scale of the instrument. The accompanying table will enable the physician to ascertain from the percentage-amount of urea in the specimen examined what is the absolute amount of that compound excreted during the day, provided, of course, the whole of the urine passed during the twenty-four hours has been collected together and carefully measured.

Per cent of urea by ureometer.	Quantity of urea in grains in 1 fluidounce.	Per cent. of urea by ureometer.	Quantity of urea in grains in 1 fluidounce.
0.1	0.456	1.9	8.658
0.2	0.911	2.0	9.114
0.3	1.367	2.1	9.570
0.4	1.823	2.2	10.025
0.5	2.279	2.3	10.481
0.6	2.734	2.4	10.937
0.7	3.190	2.5	11.393
0.8	3.646	2.6	11.848
0.9	4.101	2.7	12.304
1.0	4.557	2.8	12.760
1.1	5.013	2.9	13.215
1.2	5.468	3.0	13.671
1.3	5.924	3.1	14.127
1.4	6.380	3.2	14.582
1.5	6.836	3.3	15.038
1.6	7.291	3.4	15.494
1.7	7.747	3.5	15.950
1.8	8.203		

EXAMPLE.—The patient has passed 24 fluidounces of urine, found to contain 2.4 per cent. of urea. The total urea excreted will therefore be 10.937 (from the table) × 24 = 262.488 grains.

For exact estimations the temperature of the room in which the experiment is made must be about 70° F. (21° C.). A variation from this temperature of 20° will, however, make a difference in the result of only about 0.2 per cent., so that the temperature-correction may be regarded as unimportant.

In the process given for the manufacture of the test-solution the hypochlorite is changed into hypobromite.

This mixture gives more uniform and trustworthy results than those obtained with the chlorinated soda alone, which is recommended by Dr. Squibb. It is, in fact, identical in its action with the hypobromite solution, without the great inconvenience of handling bromine. A few minutes must be allowed to elapse after the mixture is made before mixing the urine with it; but this need occasion no delay, since the mixture can be put into the bottle before filling the cylinder and making the connections.

The activity of the solution of chlorinated soda can be easily tested by adding to a little of it in a test-tube a few drops of the solution of potassium bromide, and then a little muriate of ammonium, which should cause brisk effervescence. If this is not the case, it is too much deteriorated for use.

In some rare instances it will happen that the urine contains a larger proportion of urea than the ureometer is capable of indicating. When this is the case, and in general when the specific gravity of the urine exceeds 1.030, sugar being absent, it will be best to dilute the urine with an equal volume of water before making the test. Four c.c. of the diluted urine will then be used as usual in the experiment, but the percentage given by the reading of the instrument must be multiplied by two.

It will be found in practice that an estimation of urea by this apparatus consumes very little time, and the results for all practical purposes are as accurate as could be wished for.

Formula for Special Solution of Chlorinated Soda. Shake chlorinated lime (best quality) 12 grammes with water 100 c.c.; let settle and filter into a 250 c.c. bottle. Wash the residue with enough water to obtain 130 c.c. of clear filtrate.

Dissolve sodium carbonate 24 grammes in water 45 c.c. Add this solution to the above filtrate, mix thoroughly, and, when reaction is complete, filter, passing, if necessary, enough water through residue on filter to obtain 165 c.c. of filtrate.

The clinical value of estimating the urea is great in cases of renal disease, and it is also of value in diabetus mellitus and during pregnancy or in the puerperium and before surgical operations. The quantity of urea excreted in twenty-four hours is increased in nearly all fevers and inflammations, and is decreased in any cachectic state in which the metabolic changes in the tissues are impaired. It is also decreased in diseases which greatly modify the activity of the liver, the gland which makes urea. The particular value of estimating the urea in Bright's disease and in pregnancy lies in this, namely, that the renal disorder in these conditions results in an imperfect elimination of urea, and as a result it, or closely allied products, are retained in the blood. If in a pregnant woman or a person suffering from Bright's disease, analysis shows a constant diminution in the amount of excreted urea, the physician is warned that a uræmic poisoning is imminent, and can take active measures to relieve the patient, for, after uræmia is developed, treatment is of comparatively little value. (For estimation of urea by specific gravity, see early part of this chapter.)

Although the quantity of urea varies very greatly in perfect health, the mean amount excreted in twenty-four hours by a healthy man of twenty to forty years is about 512 grains. Women excrete a little less than men, and children still less in actual quantity, but more in proportion to their weight.

It is absolutely necessary in estimating the amount of urea excreted in twenty-four hours to test a sample of the urine obtained from all the quantity passed in that time, as a test of the urine passed on one occasion is no guide for the total daily quantity.

CHLORIDES IN THE URINE. The urine in health contains chlorides of sodium and potassium, and these are to be discovered by placing a fluidrachm of urine in a test-tube and then adding a drop of nitric acid, and finally a few drops of a solution of nitrate of silver. If chlorides are present in considerable quantity, a white precipitate of chloride of silver is thrown down, which can easily be distinguished from albumin; but if some doubt is felt as to its character, the addition of a little caustic ammonia will redissolve it if composed of chlorides, and it will be reprecipitated if nitric acid is again added. If the same quantities of urine and reagents are taken daily and placed in a test-tube of equal dimensions and the precipitate allowed to settle for twenty-four hours, we can gain an approximate estimate of the relative quantity of the chlorides. The amount ordinarily passed in twenty-four hours by a healthy man is 250 grains.

The clinical significance of a decrease in the chlorides is not great. They are decreased in the acute stages of croupous pneumonia, acute articular rheumatism, and some other fevers; and if they gradually increase, they indicate the development of convalescence.

Diazo-reaction. Ehrlich has claimed that a distinct aid to the diagnosis of enteric fever can be obtained by the so-called diazo-reaction of the urine, although it is to be remembered that this takes place in several other conditions of the body, notably pulmonary tuberculosis, measles, pyæmia, scarlet fever, and erysipelas. It is usually present only in severe cases of these ailments. Further than this, Ehrlich asserts that the reaction is usually to be obtained from the fourth to the seventh day of the disease. A faint reaction is indicative of a mild attack.

The test is as follows:

1. Take 2 grammes (30 grains) of sulphanilic acid, 50 c.c. of hydrochloric acid, and 1000 c.c. of distilled water. 2. Take a solution of sodium nitrite in water of the strength of 0.5 per cent. Fifty parts of No. 1 and one part of No. 2 solution are now placed in a test-tube and an equal amount of urine added, and this mixture is then rendered strongly alkaline by strong ammonia water. If the diazo-reaction is present, the liquid becomes carmine-red in color; and if the test-tube is shaken, this color is seen in the foam. This coloration of the foam is the point in the reaction, for, if the liquid only is red, the test is not positive. After standing a day a green precipitate will form in the tube.

The General Symptoms Associated with Urinary Disorders.

Having considered the pathological changes found in the urine and their significance, we now pass on to a consideration of the general symptoms which will usually be found associated with these variations from the normal functional activity of the urinary organs.

Let us suppose that a patient presents himself complaining that he has been seized with pain in the small of the back, and perhaps by nausea and chilly sensations, followed by a marked decrease in the quantity of urine secreted, which decrease may actually amount to suppression of the urine. The urine that is passed is high-colored or smoky in hue, sometimes looks like porter, and forms a very heavy sediment on standing. If it is filtered and tested for albumin, it will be found to contain this abnormal ingredient in large amount, and a microscopical examination of the sediment will reveal a large number of blood-corpuscles, epithelial cells, and casts (hyaline) made up of blood-cells, epithelium, and albumin. Scarcely will these signs have been noted when the patient will be seen to be anæmic, and puffiness of the face about the eyes will be evident. This puffiness may then pass on to a general anasarca, but it is to be remembered that the most violent *acute diffuse nephritis* may exist without developing anasarca. If the disease be in a child and it is due to scarlet fever, anasarca is common, as is also uræmia. The pulse in patients with this form of nephritis is usually hard and tense, and the sharp and clear sound of the heart, as heard at the second right costal cartilage, will indicate the high arterial tension. The skin is generally dry, and, it may be, harsh to the touch. Should the symptoms persist for over a month the possibility of the disease becoming chronic renders the prognosis doubtful; but, as a rule, particularly in young persons, the prognosis of acute diffuse nephritis is favorable. In the acute diffuse nephritis of pregnancy the prognosis is, of course, grave if the pregnancy continues. The history of the case prior to the attack of acute diffuse nephritis will usually be that the patient has been exposed to cold or wet, has been or is a sufferer from an acute infectious disease, has swallowed or inhaled some irritant poison, or has suffered from some severe burn of the surface of the body.

If, instead of an acute attack of illness, the symptoms just described come on gradually and insidiously, and the tendency to anasarca is marked and persistent, we have before us a case of *chronic parenchymatous nephritis*, in which the prognosis is most grave. Uræmia, vomiting, and coma may occur in this class of patients (see chapter on Vomiting). Blood-cells are also found in the sediment of the urine in these cases, but are not so numerous as in acute diffuse nephritis.

A group of symptoms which differ very markedly from those just described occur in cases of *chronic contracted kidney* (chronic interstitial nephritis). The following description of the symptoms may be taken as representing a typical case: the patient, who is usually past middle life, finds that he or she urinates more frequently and passes a greater amount of urine than heretofore. Often the sleep is disturbed by the necessity of arising to urinate. Instead of the urine being heavy and clouded, it is unusually clear and limpid; and in place of the high specific gravity of diffuse parenchymatous nephritis, we find it unusually low (only 1.010 to 1.015). Albumin is only found inconstantly and in traces, and is generally to be sought for in the urine passed by the patient when first arising from bed. The pulse is usually much increased in tension, and atheroma of the bloodvessels is more or less marked. This high-tension pulse is a valuable diagnostic sign. The heart, which in acute diffuse nephritis may be slightly dilated, or in chronic parenchymatous nephritis somewhat hypertrophied, is in this disease usually markedly hypertrophied, and the second sound at the second right costal cartilage is commonly accentuated. In addition to these symptoms we find that chronic bronchitis is not rare, and that pulmonary œdema and attacks of shortness of breath which may be called asthmatic, are often present, the latter being most marked at night. Uræmic symptoms are more commonly seen in this class of cases than in any other, and violent vomiting difficult of control should always make the physician test the urine to discover renal mischief. Unlike parenchymatous nephritis, dropsy is a rare complication of chronic contracted kidney. Microscopic examination of the urine will reveal only a few hyaline and granular casts.

The prognosis as to cure is bad, but life may be prolonged indefinitely.

A copious flow of urine of a low specific gravity and of a pale, clear appearance, containing fatty, hyaline, and finely granular casts, is often seen in cases of *amyloid disease* of the kidney, and the presence of syphilis, of prolonged suppuration, or extensive bone disease, due, it may be, to tuberculosis, with concomitant enlargement of the liver and spleen, separates it from any other ailment. Albuminuria may be a marked or an absent symptom.

Let us suppose, however, that a patient comes to us with a history of exceedingly copious urination, of great thirst, of loss of flesh, and has a dry, harsh skin, we immediately recognize that a test of the urine will probably reveal the case to be one of *diabetes mellitus.* This will be pointed to if a high specific gravity is found present in a clear limpid urine, and confirmed if the tests for sugar already given produce a reaction. The other prominent symptoms of diabetes mellitus are furunculosis, intense itching and erythema (see chapter on the Skin), an excessive appetite, and, in some severe cases, gangrene of the extremities or diabetic coma (see chapter on Coma and Unconsciousness). If the urine has a constant low specific gravity and contains no albumin or sugar, the case is probably one of diabetes insipidus.

Should much pus be present in the urine, it is probably derived from a *pyelitis* or a suppurative inflammation of the pelvis of the kidney. The symptoms of this state are, briefly, a constant or intermittent pyuria, usually an acid reaction of the urine, chills and fever, which may mislead the physician into a diagnosis of malarial poisoning, or, in other cases, if the pyelitis be tubercular, hectic fever may be present. Sometimes violent attacks of pain resembling renal colic are passing symptoms, and not uncommonly anæmia and loss of strength are notable. There is often pain in the back, which is made worse by pressure with the hand, and, rarely, if the suppurative process be marked, typhoid symptoms may be present.

If the pyelitis be tubercular, tubercle bacilli may be found in the urine. If due to a calculus, there may be a history of gravel and renal colic. The purulent urine of pyelitis is to be separated from that of cystitis by the fact that in the former the urine is acid, in cystitis it is ammoniacal. Additional aids to the diagnosis are the pain in the renal region, often unilateral; and the use of the cystoscope to exclude vesical disease. The prognosis varies. If due to an infectious fever, recovery usually occurs. The patient may also recover from tuberculous pyelitis.

CHAPTER XII.

THE BOWELS AND FECES.[1]

Constipation and diarrhœa—The cause of these two symptoms and their diagnosis—The diseases in which these symptoms occur—Choleraic diarrhœa—Dysentery—The color of the feces—Intestinal parasites.

THE consideration of the condition of the bowels and feces as indicative of disease affecting the intestines themselves and other organs closely associated with their functions can be best divided into several parts, namely, the functional disorders of the intestines and the organic diseases from which they may suffer, on the one hand, and the appearance of the feces in both functional and organic diseases of the abdominal viscera in general, on the other. The most common forms of intestinal disturbance are constipation and diarrhœa.

CONSTIPATION may be due to mere sluggishness of bowel-movement because of both nervous and muscular atony, or to deficient secretion of the intestinal juices, or, again, to the too rapid absorption of the liquids from the fecal matter while it is passing through the colon. It is also associated with all those conditions which prevent the proper secretion of bile, which liquid very materially increases peristalsis. Thus, we see obstinate constipation in most cases of true jaundice; in cases of hepatic disease, producing a deficient biliary flow; and in phosphorus-poisoning, in which the fatty degeneration and hepatitis prevent biliary secretion. Further than this, the constant ingestion of foods which are absorbed nearly *in toto*, or, in other words, leave little residue, particularly raw or boiled milk, produces constipation. Again, the use of wines containing large amounts of tannic acid may produce similar results because of the astringency of this substance, and chronic constipation from the use of large quantities of badly infused or boiled tea made with hard water is frequently met with. When the feces are very dry, the cause may be lack of liquid ingested, and the remedy be full draughts of pure water; or, again, constipation occurs as a manifestation of diabetes insipidus or diabetes mellitus, because the polyuria characteristic of these affections drains the body of liquid. Obstinate constipation should, therefore, always call the physician's attention to these affections and to two other possibilities, namely, that the condition depends upon wilful disregard by the patient of the calls of nature, so that the bowel is forced to retain fecal matter until it becomes hard and dry; or, quite as important, that the constipation may be due to some reflex cause, which, as the result of irritation, results in an arrest of peristaltic movement. Thus, a woman with ovarian and other pelvic trouble may have obstinate constipation which yields little, if at all, to purgatives, but readily to nervous sedatives or even to an opiate. Or, again, in chronic lead-poisoning the inhibitory fibres of the splanchnic nerves may be so irritated that peristalsis is impossible. Here a hypodermic injection of morphine may make a movement possible.

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[1] For intestinal obstruction in its various forms see chapter on Vomiting.

The organic diseases of the bowel producing constipation are many and of great importance. They consist in intestinal obstruction in all its forms, as by bands, growths, by the process of intussusception, by volvulus, by cicatricial contractions, and by impacted foreign bodies or fecal matter. The presence of a sudden attack of constipation, or the presence of this condition in a degree which fails to yield to mild laxatives, should always put the physician on his guard lest some such grave condition is present. As severe and, finally, stercoraceous vomiting is a fairly constant and more marked symptom of intestinal obstruction than is constipation, a discussion of the various symptoms of intestinal obstruction will be found in the chapter on Vomiting, and the diagnosis of growths of the intestine will be found in the chapter on the Abdomen.

Aside from these causes, it is manifestly impossible to discuss all the conditions of the system in which constipation may be present. The physician must always bear in mind that constipation often results in the absorption of poisonous materials from the bowels, which in turn may produce all sorts of symptoms, nervous or otherwise, from epileptiform attacks, in rare cases, to severe headache and vertigo, with vomiting, in others.

DIARRHŒA of an acute type depends, as a rule, upon one of four causes, namely, the presence of irritant material in the bowel, which the intestines attempt to get rid of by increased secretion and excessive peristalsis; relaxation of the blood-vessels of the intestine, with profuse serous leakage and consequent watery purging; acute inflammation, with excessive secretion of mucus; and the endeavor of the system to eliminate poisons in this manner, as in cases of sudden profuse diarrhœa, in cases of chronic renal disease, in which the purging is an effort at elimination. The last-named forms of diarrhœa are usually sudden in onset and speedily get well of themselves, and it is a mistake to check them too suddenly.

It is impossible to speak of all the possible causes of diarrhœa, or of all the diseases in which it is met with. Only those in which it is a prominent symptom, or one of importance, can be discussed.

One of these is cholera morbus, a disease which manifests itself in profuse watery purging, accompanied by violent pain in the belly, and, after several stools have passed, in a considerable amount of tenesmus. Mucus is almost entirely absent from the dejecta, but particles of undigested food may be found in them. Vomiting is often a severe and simultaneous manifestation of the gastro-intestinal disorder which exists, and, if the attack be very severe, it is practically impossible to separate it from true cholera Asiatica if an epidemic of that disease is present. The patient speedily becomes cold and pinched-looking, exceedingly weak, and finally passes into collapse. The pulse becomes feeble, rapid, and running; the face livid, and finally the patient may develop the *facies Hippocratica.* The urine is greatly decreased or entirely suppressed, because of the watery purging, and possibly by reason of the effects of certain poisons upon the kidneys. In the great majority of cases the symptoms are not so severe as this, and complete recovery ensues as soon as the offending materials are passed out of the bowels and the patient has time to convalesce.

When an attack of diarrhœa, such as has just been described, comes on in a young child it is usually called cholera infantum, or summer complaint, and it is nearly always due to improper feeding or to the unintentional use of bad food or bad milk. The stools of the child are usually at first filled with curds of milk and green masses, looking as if the curds had been stained with grass-juice or spinach. The child often passes with extraordinary rapidity into a state of collapse, and may die in a few hours or days. The tenesmus often becomes constant and is a distressing symptom, and the tissues become shrunken to a marked degree. The child manifests not only the evidences of the results of profuse purgation, but, in addition, is evidently intoxicated by the toxins absorbed from the bowel, so that it lies on the lap of the nurse in a relaxed and torpid state. The surface of its body is often abnormally cold, and its extremities may be pinched and blue; but the temperature of the internal organs is generally abnormally high, so that while the axillary temperature may be below normal, the thermometer will reveal a temperature of from 102° to 103° in the rectum. Sometimes the head becomes retracted, as if meningitis was present. The respirations may be sighing or of the Cheyne-Stokes type.

If the child or adult is seized with symptoms such as those described under *cholera morbus* or cholera infantum, and a suspicion of the presence of true cholera is raised, are there any facts which will point to the correct decision in a case, even if, as already stated, a positive differential diagnosis cannot be made? In the first place, a train of symptoms of a malignant type points to the true cholera, rather than cholera morbus, or cholera nostras, as it is sometimes called. Again, the evidences of infection or general systemic disease indicate the epidemic malady rather than does a profuse diarrhœa alone. Thus the systemic signs of infection may be so great that death from infection in true cholera occurs before diarrhœa even begins. Again, it would be possible to determine the presence of true cholera if the comma-bacillus could be demonstrated; but this requires the examination of the fecal matter to be made by an expert who is familiar with the technique of examining fecal matter for the germs and with the necessary measures for their artificial culture.

Symptoms identical with the more violent forms of cholera nostras or true cholera may be produced by *acute poisoning by antimony*, except that in this case we often have profuse sweating and salivation early in the attack. The same symptoms of vomiting, purging of rice-water stools, collapse, cramps in the calves of the legs, and violent pain in the abdomen may be present. A differential diagnosis without the history of the patient having taken poison is impossible, except by a chemical analysis of the vomited matter, which will contain antimony, as will the stools and the urine. The utmost care should be used that the vessels which receive these materials are chemically clean, that they are hermetically sealed until ready for the expert analysis, and that they are in the hands of thoroughly responsible parties up to the date of analysis.

While *arsenic* may cause somewhat similar symptoms to those due to antimony, the stools are generally bloody from destruction of the gastro-intestinal mucous membrane by the drug. Rarely certain poisonous toadstools produce somewhat similar symptoms.

If an adult who has not eaten anything which could have produced a diarrhœa, as the result of irritation from bad food, is seized with profuse watery purging, with very little or no pain, and without nausea and vomiting, it is probable that he is suffering from the *acute nervous diarrhœa* which sometimes results from exposure to severe nervous strain. To illustrate the character of these cases the author may mention the fact that it is quite common for him to see medical students, exhausted by a long winter's work and anxious about their examinations, seized by an attack of profuse watery purging in the middle of the night preceding the examination of which they stand most in dread.

Care must be taken by the physician in all cases of sudden and profuse diarrhœa to which he is called to exclude the presence of renal disease, for purging may be an effort at elimination of effete materials, and its sudden arrest by drugs may induce uræmic convulsions or coma.

Sudden attacks of profuse watery diarrhœa in which the patient passes great quantities of liquid from the bowel, with or without pain in association therewith, may be due to *locomotor ataxia*, manifesting itself in an "intestinal crisis."

In cases of persistent or obstinate diarrhœa, serous or catarrhal, in which there is an excessive peristalsis which hurries the intestinal contents along so fast that the food cannot be properly digested, the physician should remember that *fissure of the anus* or some other source of irritation may be present in the lower bowel which produces reflex excitability of the nerves governing the bowel-movements. In other cases a stricture in a feeble, dilated rectum will cause retention of feces until irritation, tenesmus, and even loose mucous movements are produced.

If, instead of watery or serous movements, the patient is attacked by a more or less acute diarrhœa, accompanied by great pain and distention of the belly, and if there is marked tenderness on pressure over the transverse colon and mucus in the feces, which are not in very large quantities after the first few movements, there is probably present the condition known as *entero-colitis*, or inflammation of the ileum and colon. It is met with in both children and adults, and differs in its course from cholera morbus and cholera infantum very markedly. The pain is usually more constant, more aching, and less griping in character. Vomiting is not a constant feature, as it is in the watery choleraic diarrhœas, and the course is more subacute, the duration of the illness usually being from one to three weeks. If food which is difficult of digestion has been eaten, it is passed, still undigested, from the bowel, and is apt to be coated with mucus. Such a diarrhœa is called *lienteric diarrhœa.*

Not far removed from this type of cases are those of a more chronic character depending upon more grave and lasting alterations in the gastro-intestinal mucosa. As a rule, the greater part of the trouble exists in the colon, and more or less griping pain in the neighborhood, namely in the upper umbilical area and left groin, may be present before each movement. The abdomen is apt to be distended and quite tender on pressure, particularly in varying spots, and considerable loss of bodily weight is apt to ensue, chiefly from failure on the part of the digestive tube to absorb the food that is eaten. The movements are not markedly watery, but are usually unformed and about the consistency of oatmeal gruel or a little thicker. Flakes of mucus are often found in large amounts in the fecal matter, and the feces may be frothy or flaky as the result of fermentation. Blood and pus are very rarely seen in the movements of these cases, unless the blood escapes from an inflamed hemorrhoid. Sometimes, when these cases are very severe in character, the mucus takes the shape of long cord-like or worm-like strings, or even seems to be membranous in character. In other instances the feces, when formed, are passed in ribbon-shaped masses, due either to spasm of the muscular fibres of part of the lower bowel or to cicatricial contractions from the healing of old ulcerations. In very severe cases the condition of the intestines gradually advances from a *mild follicular entero-colitis* to one of actual deep ulceration, and under these circumstances blood and pus may be present in the movements. At such times the pain produced by the patient having a movement of the bowels, or by the passage of fecal matter over the ulcerated surface, may be intense, and the invalid will often state that the pain feels as if one spot in the gut were made more painful by the feces rubbing over it. Such cases often continue for years, while some of them ultimately get well, others become chronic invalids from the slow changes in the intestinal walls. In this connection the diarrhœa of *tuberculosis* is not to be forgotten, depending, as it does, either upon the general infection or upon the development of ulcerations in the intestinal canal.

In some cases in which the patient after exposure to cold or wet is seized with violent pain in the epigastrium and a feeling of weight in the rectum, a few loose movements and then intense tenesmus and bearing-down, with only a few drops of mucus in the way of a movement, the condition is one of acute rectal catarrh or *proctitis*.

The cases just named in the preceding paragraphs are to be separated from those in which there is *true dysentery*. Dysentery is a term very loosely applied, by the laity in particular, to any form of severe diarrhœa, particularly if there are blood and mucus in the movement. In reality the term dysentery should be limited to cases due to an infection and very apt to occur in epidemics. As Osler says, true dysentery is one of the four great epidemic diseases of the world.

Let us suppose that a patient is seized with diarrhœa and some pain in the belly, and with only a slight chill, or this symptom may not be present. The pain soon becomes more and more colicky, and the stools are passed with ever-increasing bearing-down or tenesmus. The effort to empty the bowel, after it is in reality thoroughly emptied, results in agonizing bearing-down pains. Fever to the extent of from one to three degrees may be present. Thirst is excessive, the stomach is usually retentive, and the stools are first the ordinary bowel-contents, and then mucus, which may be blood-streaked. Soon the mucus becomes jelly-like in appearance and more thick and tenacious, and, finally, after several days it begins to look muco-purulent, and the stools are less frequent. Sometimes small, bullet-like, hard pieces of fecal matter are shot out of the rectum after severe straining. Recovery usually begins at from seven to ten days. The entire trouble seems to be in the large bowel, and particularly in the sigmoid flexure and rectum. Such are the symptoms of ordinary mild dysentery of hot climates or of summer weather in the temperate zone.

The severity of the disease is much greater in hot weather, and the prognosis is not good in severe cases coming on during an epidemic.

On the other hand, if the patient has an irregular diarrhœa after or during a residence in tropical parts, which may or may not have a sudden onset, with moderate fever and considerable loss of flesh, and has moderate bellyache, which soon becomes much less, and if the stools as just described above become more and more fluid, and the diarrhœa intermits, the physician should think of the case being probably one of so-called tropical dysentery, or *amœbic dysentery*, a condition of infection by the so-called amœbæ coli. The course of the disease is slow, lasting from six to twelve weeks, and the death-rate is high. Convalescence is always very slow, and liver-abscess due to an hepatic infection by the amœbæ coli is very frequent. Sometimes secondary abscess of the lung develops.

A positive diagnosis of this variety of dysentery is made by the discovery of the amœbæ in the stools. These micro-organisms possess active amœboid movements and are found in greater number when the diarrhœa is severe. They are to be sought for in the small gelatinous masses which are found in the feces. Sometimes the entire stool seems loaded with amœbæ; at other times only a most careful search will discover them. They are more refractive than the cells found in the feces, and contain numerous vacuoles, so numerous in some cases that the cells look very granular. These must not be mistaken for the compound granular bodies found in the feces. When they are active a division into an endosarc and an ectosarc can be discovered. Often red blood-cells will be found in the amœbæ. Epidemic dysentery is usually due to Shiga's bacillus.

Sometimes a diphtheritic or pseudomembranous dysentery is developed in persons having chronic heart disease, and it has been seen as a sequel of acute croupous pneumonia. This is called *secondary diphtheritic dysentery*, and death generally results from exhaustion, only a suspicion of the intestinal condition having existed during life. Such a state is sometimes a complication of Bright's disease, probably owing to the irritation of the intestinal mucous membrane produced by the urea decomposing into the carbonate of ammonium. In *acute primary dysentery of a diphtheritic character* the patient may rapidly pass into a typhoid state, and the case be diagnosed as one of typhoid fever with profuse diarrhœa. The discharges are the only means of separating the two conditions (enteric fever and diphtheritic dysentery), as they often are filled with blood and mucus in dysentery, a condition rarely seen in typhoid fever.

Dysentery may be confused with the diarrhœa sometimes produced by a malignant and ulcerating growth in the sigmoid flexure or rectum, but a physical examination will usually reveal the tumor, and the cachexia will aid in pointing to it as the cause.

Syphilitic ulceration of these parts may cause a somewhat similar train of symptoms. Again, it is by no means rare to meet with the passage of several muco-purulent movements each day in persons who have pulmonary gangrene or pulmonary tuberculosis, partly due to the swallowing of fetid sputum or tubercular ulceration of the bowels. Diarrhœa is also a symptom of septicæmia. Distantly allied to this form of diarrhœa is that seen in persons who have dissected a putrid body ("dissecting-room diarrhœa," so called).

While it does not fall to the lot of this book to discuss the symptoms of the affections of the rectum which are to be relieved by surgery, it is proper to speak of the causes of *blood in the stools* in other states than dysentery. In inquiring as to the blood in the stools, we should ask whether it is mixed with the feces or is seen in streaks, and whether it passes in jets or not. If in jets, it will be found in the pan away from the fecal matter. We should ask as to the amount of blood and its color. If mixed with the feces, it probably results from a slow oozing from a hemorrhoid, or, if the feces are formed, from some leaking vessel in an ulcer high up in the sigmoid flexure. If in streaks, it probably comes from the wall of an ulcer which has been scraped by the fecal mass. If it is passed in jets, it probably comes from some vascular but small arterial tuft of vessels low down near the anus; and, finally, if it is dark and tarry-looking, it is probably due to a leaking in the upper colon or ileum ; whereas if light red in color it is from vessels in the rectum. Most commonly it is from hemorrhoids, or from an ulcerated mucous membrane covering a syphilitic deposit, or else a malignant growth. (See further on in this chapter.)

Finally, it is interesting to note that paroxysmal attacks of seromucous or bloody diarrhœa sometimes come on in cases of exophthalmic goitre. Diarrhœa of a more or less severe type may come on in cases of hysteria, often associated with tremendous eructations of gas and rumbling in the stomach and bowels.

Fatty diarrhœa may ensue if feeble persons already suffering from irritable bowels take an excess of cod-liver oil, but in other cases it possesses great diagnostic importance. If associated with diabetes, it gives us reason to believe that there is some disease of the pancreas producing both the glycosuria and the lack of digestion of the fats. Sometimes in jaundice, however, fat is found in the stools owing to the lack of bile to emulsify it in the intestine.

The Feces. In this connection we naturally pass on to a discussion of the diagnostic indications of the feces. In the first place, it must be remembered that the quantity of the feces depends upon the quantity of the food, and again that the quantity varies with the character of the food, for if the food be such as to be bulky, yet contains little nutritive material, there will be a large residue to be passed out in the feces; whereas if the food be almost entirely composed of materials which can be assimilated very little residue is left, and the feces are consequently smaller in bulk. Thus, the cow eats a large bulk of food and passes large amounts of fecal matter, whereas the dog eats meat and passes very small amounts of fecal matter.

Again, it is not to be forgotten that many foods actually increase intestinal peristalsis, and so produce large and loose movements, as oatmeal and wheaten grits or apples, while other foods, such as cheese, do the opposite. If the stools are large and copious and the food which the patient has taken is in reality not of a kind leaving a large residue in the bowel, the indication is that there is non-absorption of nutritive materials, with probable wasting of the patient.

The consistency of the feces in health varies from a formed "stool" to a mushy condition; but in disease we have a liquid watery stool if the trouble be serous diarrhœa, and a pasty and slimy stool if it be due to a catarrhal state of the bowels. The passage of hard scybalous masses mixed with liquid indicates that the feces have become dried and hard in the sacculations of the colon, and are passed out only when they cause so much irritation as to produce diarrhœa. If the feces are in narrow bands or flattened ribbon-shapes, there is probably a stricture of the rectum, offering an obstruction to their passage. A mushy or semi-watery stool is often seen in typhoid fever.

The odor of the stools depends very largely upon the food which is taken and upon the degree of fermentation present in the bowels. In nursing children the stools often have a faintly sour odor, and in the diarrhœa of nurslings with acid fermentation there is an odor of the fatty acids. If the process is marked, this odor becomes actually foul, and in cholera infantum the stools have a musty, mousy odor. If malignant growth of the bowel is present, the odor is fetid, as it is also in gangrene of the intestine. Sulphur when taken internally sometimes causes a very offensive stool, owing to the sulphuretted hydrogen gas which is developed in the bowel.

The color of the stools is of great diagnostic importance in several conditions. In health the feces should be brown or brownish-black, the color being partly due to the food, but chiefly to the bile (hydrobilirubin). Certain fruits render the stools dark in color, and drugs, such as iron and bismuth, do likewise, and hæmatoxylon often makes them look red.

In the stools of persons living on a pure milk-diet we usually find little color comparatively. Again, in cases of jaundice, phosphorus-poisoning, and acute yellow atrophy of the liver, the stools are very light in color, owing to their lack of biliary coloring. They are also apt to be very light in chronic lead-poisoning.

Bilious stools are either golden-yellow, greenish, or reddish in hue, and if the flow of bile is profuse, they are apt to be watery. Greenish stools looking as if they contained chopped spinach are, however, a peculiarity of the diarrhœa of fermentation, particularly in infants, the color being due to color-forming micro-organims; but a greenish stool may also be produced in an infant by the persistent administration of sodium bicarbonate.

If the stools are well mixed with mucus, the catarrhal process probably exists in the ileum; but if they consist of hard masses of feces coated with mucus, the disease is probably a colitis.

Bloody stools are most commonly due to hemorrhoids which are eroded. The blood may be bright if the hemorrhoid be a small arterial bunch, or more dark and grumous if slow oozing has gone on for some time prior to the movement. As a rule, the brighter the blood in the stool the nearer its source is to the anus, and the darker the blood the higher is its source in the bowel. Thus, if the stools are tarry-looking, the blood is almost certainly from the small intestine, and probably arises from a duodenal or other ulcer or from carcinoma of the stomach or bowel; while if it is only somewhat changed in appearance, it may be due to an ulcer or ulcerated morbid growth in the colon. Sometimes, however, where the hemorrhage from the ileum is very profuse, as in typhoid fever, the blood comes from the anus only slightly changed in appearance. If blood is suspected to be present, we can confirm its presence by finding the corpuscles with the microscope or by testing the feces for hæmin. (See chapter on Vomiting.)

Stools containing pus may receive this material from the surfaces of ulcers, but usually the source of the purulent matter, if it is present in large amount, is an abscess which has ruptured into the bowel, as, for example, in peri-rectal abscess.

Finally, we may find gallstones in the stools, which, if they are passed soon after their escape into the bowel, are found to be faceted. Stools which are being searched for gallstones should be washed through a sieve in such a way as to catch the stone and let the fecal matter through. The intrahepatic gallstone is not faceted and crumbles easily. This rarely escapes, because it is imbedded, and if it does get into the bowel is usually broken up. All stones or concretions found in the feces are not to be considered as gallstones. They may be pancreatic calculi in rare instances, or they may be fecal stones (coproliths) or intestinal stones (enteroliths). Fecal stones are simply hard inspissated masses of feces, which may attain a very large size, whereas intestinal stones are composed of heavy, brown concentric layers of phosphates of calcium and magnesium around some nidus, as a seed or piece of bone. Sometimes they are concretions of insoluble drugs, such as salol or magnesium carbonate.

Very rarely a portion of the bowel sloughs away, and yet recovery takes place. This is seen sometimes in intussusception.

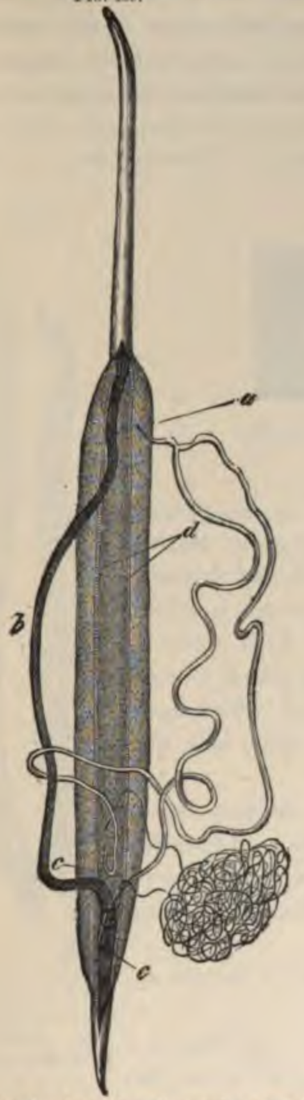
Aside from the character of the stools themselves, we often search for the cause of an ailment in the passages, either for foreign bodies, such as pebbles or pins, or for *intestinal parasites* (worms). Sometimes worms may exist for long periods of time in the bowel without causing any symptoms, and, again, in children in particular, they cause great systemic disturbance by producing disorder of the digestion or reflex irritation.

Worms. Under the name of tapeworm or cestodes we find in the intestine, and often in the stools, a parasite occurring in segments which are flat and ribbon-like, and usually from a quarter to one-half inch in length. The worm itself may be several yards long. Its head is small, and it maintains its hold on the bowel by its head. The segments are usually broken off one by one and escape in the stools, and the stools also contain the ova or eggs of the parasite, which are developed in each segment, which also possesses male and female organs.

According to the shape of the head and the size of the worm and the source of infection, we divide tapeworms into three classes: the tænia solium, the tænia mediocanellata, and the bothriocephalus latus.

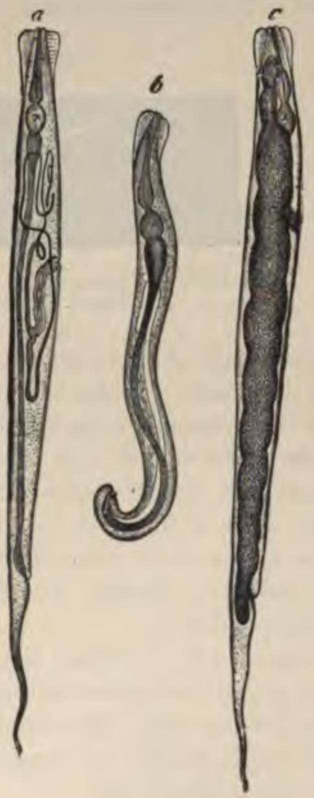
If the patient passes a worm of from one to three yards in length, the head of which is about the size of a pin-head and glistening gray in appearance, the rest of the worm being yellowish-white, and if upon the head can be seen four pigmented suckers surrounded by a crown of hooks, that worm is a tænia solium, and is probably derived by the patient from raw or uncooked pork. The eggs of the tænia solium must be sought for by a microscope. They are round and covered by a hard shell, which upon pressure breaks into small fragments. In the shells may be found a few hooklets. These eggs are passed out in the feces by the host, and are then swallowed by the pig, in whose muscles the hooklets migrate and form cysts. In these cysts the hooklets develop, and when a man eats the meat raw they enter his intestine, attach themselves, and from them a tapeworm is developed.

FIG. 189.



Ascaris lumbricoides, dissected and walls thrown back. (HELLER.) *a*. Genital orifice. *b*. Intestine. *c*. Oviducts. *d*. Longitudinal band. *e*. Ovaries.

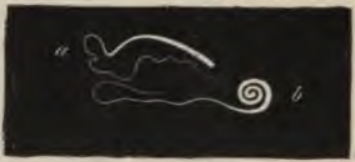
FIG. 190.



Oxyuris vermicularis, magnified. (PAYNE.) *a*. Young female. *b*. Male. *c*. Mature female, full of eggs.

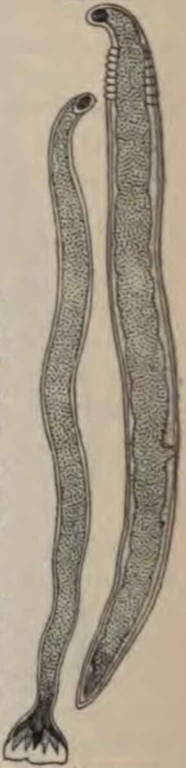
If the worm is from four to five yards long and the segments after leaving the anus have motile powers, and if the head is larger than that of tænia solium and devoid of hooklets about the suckers on its head, it is probably the tænia mediocanellata or saginata. The egg is slightly larger than that of the solium. This worm usually comes from eating raw beef. The bothriocephalus latus is the largest of all tapeworms, often reaching seven to eight yards in length. It has a long head with two long, narrow suckers. The eggs are oval, very large, and the shell is light brown in color, and very easily broken. This parasite is not common in America, but is a very frequent cause of profound anæmia in the persons whom it infects. Its joints are only rarely thrown off, so its presence is often overlooked, and this renders the search for the eggs very important in severe anæmia with no ascribable cause. This worm is usually derived from fish. A worm which is comparatively rare is the *tænia cucumerina*, which has a head with sixty hooks. It infects dogs, cats, and sometimes children.

FIG. 191.



Trichocephalus dispar, natural size. (PAYNE.)
a. Female. b. Male.

FIG. 192.



Ankylostomum duodenale, magnified. (BRISTOWE.)
a. Female. b. Male.

A round worm, looking like an ordinary earthworm, appears sometimes in the stools, and is called ascaris lumbricoides. It is sometimes vomited, and, rarely, causes trouble by crawling into and blocking the common biliary duct.

Fine thread-like worms inhabiting the rectum are the oxyuris vermicularis.

A very important diagnostic find in the feces is a worm looking very much like the thread-worm, but somewhat larger, which inhabits the duodenum. It is called the ankylostomum duodenale. The importance of finding it lies in the fact that it produces the most profound and acute anæmia by sucking blood from the intestinal wall. The worms are usually only found after a vermifuge is taken, but the eggs are always present in the feces as unsymmetrical, thickly covered, segmented globules. If the stools containing the eggs be set aside in a warm place, the embryos can be seen to develop. Bloody stools may be due to the presence of this parasite. The so-called whip-worm, or trichocephalus dispar, is a fine thread-worm without any medical interest.

CHAPTER XIII.

THE EYE.

The general diagnostic indications afforded by the eye—Diplopia and disorder of the external ocular muscles—Strabismus and squint—Disorder of the internal ocular muscles—The pupil—Hemianopsia—The visual fields—Color-vision—The optic nerve and its lesions—Retinitis—Amblyopia and blindness.

THE eye affords more information for diagnostic purposes concerning the condition of other organs of the body than any single part which can be examined. We gather from it not only a clear idea as to its own state, and the state of the nervous centres more or less intimately connected with the government of its movements and its special functions, but in addition we often gain positive information as to the condition of organs more remotely situated, as, for example, the kidneys. The very fact that so many different tissues are found in this organ renders it susceptible to the many diseases affecting similar tissues elsewhere in the body. The parts of the eye which give us the greatest amount of knowledge about changes in other tissues are the optic nerve and retina and the ocular muscles. The crystalline lens, the conjunctiva, and the cornea often give additional evidence indicating the general systemic condition. Cataract should make the physician suspect diabetes, even if it appear in persons advanced in years. The eyelids, if puffy in appearance, may indicate renal disease, cardiac lesions, or the over-use of arsenic (see chapter on the Face). An examination of the inner side of the lids may reveal a pallor due to anæmia. Slight conjunctival hemorrhage may result from violent coughing. In old persons such a hemorrhage, if not due to injury, may indicate degenerative vascular changes.

Prominence of the eyeball, or exophthalmos, is seen as an almost constant symptom of true goitre, which for this reason is called exophthalmic goitre. (See Figs. 11 and 12.) Associated with the bulging eyeball we find more or less enlargement of the thyroid gland, an irritable heart, and a very rapid pulse, throbbing carotid arteries, marked general nervousness, often mental depression, and insomnia. In well-marked or advanced cases of exophthalmic goitre we often have a condition in which the upper eyelid does not follow the eyeball in its downward movement. This is sometimes called "Graefe's symptom." Again, there may be almost total absence of winking as an involuntary act, "Stellwag's symptom." Or, again, there is insufficiency of convergence, so that a near point cannot be seen with both eyes at once (Moebius's sign).

On examining the exterior of the eyeball we often notice a grayish ring along the junction of the cornea and sclera. It possesses when a complete ring but little significance, except age; but if it is the segment of a ring or in two segments, at the upper and lower margin of the cornea, it is a true *arcus senilis*, and is said to indicate in some cases fatty degeneration of the tissues of the body. The one is an annulus senilis, the other an arcus senilis, and the arcus is the change worthy of note, although many clinicians, including the author, deny that either has any significance.

An examination of the pupil may reveal that it is immobile from an old plastic iritis, due to syphilis or rheumatism, but it is not to be forgotten that this condition may arise from iritis due to purely local causes. A widely dilated pupil may indicate the use of some mydriatic or the ingestion of atropine. Such a pupil is also seen in fright, in some hysterical seizures, and in glaucoma and whenever the vision is lost, unless the pupil be contracted by disease of the iris. A contracted pupil indicates the use of a myotic or the existence of central nervous disease, such as ataxia, which causes the Argyll-Robertson pupil as well. Sometimes corneal inflammation by causing photophobia may cause excessive myosis. Pin-point pupils may also result from the use of opium or its alkaloids, and serve to differentiate the condition from true coma, in which the pupils are usually dilated. If, however, the coma be due to cerebral inflammation or meningitis, the pupils may be contracted; or if it be due to intracranial pressure, they are usually dilated. (See Paralysis of the Intraocular Muscles.)

In addition to these objective symptoms we have also a very important set of signs connected with the ocular muscles, external and internal, as manifested by the various forms of strabismus or changes in the pupil and in the accommodation of the eye, by the ptosis already discussed in the chapter on the face, and in nystagmus and ocular spasm. Beyond this, too, we have two other ocular symptoms subjective in nature, namely, diplopia, or double vision, and partial or complete blindness.

Diplopia depends upon the fact that in an eye in which the muscles are abnormal in their function the image which falls upon the fovea, or visual acuity spot of the retina, in the well eye fails to fall upon the same spot in the weak eye. To the well eye the object appears to be in the direction in which the eye is turned, whereas to the weak eye it appears to be in another direction. As a result, the mind gets the impression of two objects instead of one. The impression made on the well side is the "true image," as it is called, and that in the diseased eye is called the "false image." Any cause which interferes with the fixation of each eye on the same point produces dipolpia, and, as the eyes are normally directed to the object fixed by the ocular muscles, paralysis of any one of these muscles produces diplopia when the axis of one eye is deviated from the point of fixation, because the eye on one side is not properly moved by reason of the fact that one muscle has failed. Diplopia is ordinarily a constant sign of ocular muscular paralysis; but if only weakness or insufficiency of a muscle is present, diplopia may never be a symptom recognized by the patient. The forms of diplopia—that is, the position of the false images in respect to the true images—vary with the muscles affected, and will be studied in a moment when paralysis of the muscles is tested for and their diagnosis discussed. It only remains at this place, therefore, to point out the probable significance if a patient with diplopia presents himself to a physician.

Thus, a patient with diplopia may be suffering from a lesion in the cerebral cortex, such as hemorrhage, sclerosis, or softening; or from a lesion in the cranial nerve nuclei, in the pons or corpora quadrigemina, or in the fascicular fibres. Again, diplopia may arise from lesions at the base of the brain, as meningitis, tubercular or syphilitic, or from injury to the nerves in the orbit or in their peripheral endings. As a result, we find diplopia as a symptom of any disease which may affect these parts, and it is quite a common symptom in locomotor ataxia, in Friedreich's ataxia, and in paretic dementia. Probably it is seen most commonly in ataxia, and with it, as the oculomotor nerve in its branch supplying the levator palpebræ is particularly apt to be paralyzed in this disease, we may find ptosis.

Diplopia is also found in cases of ptomaïne-poisoning, and in poisoning by belladonna, spigelia, conium, and gelsemium, owing to their effects on the ocular nerves.

The differential diagnosis between the various lesions producing diplopia is to be made by the other symptoms and the history of the case.

Paralysis of Ocular Muscles. As something has already been said in the chapter on the Face and Head of the diagnostic import of paralysis of the ocular muscles in connection with the subject of ptosis, a further consideration of the abnormal changes in their functions will be discussed first in the present chapter.[1] Before doing so, however, it is necessary to describe the methods resorted to for the purpose of demonstrating or determining departures from the normal in these muscles. In the first place, it must be clearly understood that the function of the extrinsic muscles of the eyeball is to direct the ball toward the object at which the patient desires to look, and they also evenly balance one another to keep the eye steady in its axis. Thus, the external and internal rectus muscles maintain the horizontal equilibrium of the eyeball. If the internal rectus is completely paralyzed in one eye, we have developed a unilateral external squint, the eye looking toward the outer side of the orbit; and if the external rectus fails, the eyeball is turned toward the nose. If these muscles are affected in both eyes, we have a divergent squint in the first case and a convergent squint in the second. Not only do the muscles of each eyeball govern the eye-movements of that side, but by the nervous centres governing the eye-muscles the two sets of eye-muscles are co-ordinated, so that they move as one organ in health.

Just here it is well for the reader to make a clear distinction between concomitant and paralytic squint, for they are two very different things in origin, symptoms, course, and prognosis. A concomitant squint is a wrong relation in the visual axes, so that they do not intersect in the point looked at; but there is no marked limitation of the movements of either eye in any direction. Be the direction of the eyes what it may, the squint remains practically unchanged. Further, if the fixing eye is covered, the other eye promptly fixes, and the covered eye deviates without the patient altering the position of the eye (Jackson). On the other hand, paralytic squint is the deviation which takes place when the attempt is made to turn the eyes in certain directions by means of the muscles which are paralyzed in whole or in part. When the attempt is made, the eye with the sound muscles turns as it should, while the eye with a paralyzed muscle hangs back, beginning to deviate as the eyes are turned, so that this muscle is required to perform its function, and deviates more as greater effort is required. The degree of squint and of separation of the double images it causes varies with the direction in which the eyes are turned, there being none at all in certain directions.

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[1] In the preparation of this chapter free use has been made of the article of my friend, Dr. de Schweinitz, on "Diseases of the Cranial Nerves," in Dercum's "Nervous Diseases."

We examine the functional activity of the ocular muscles by the following measures:

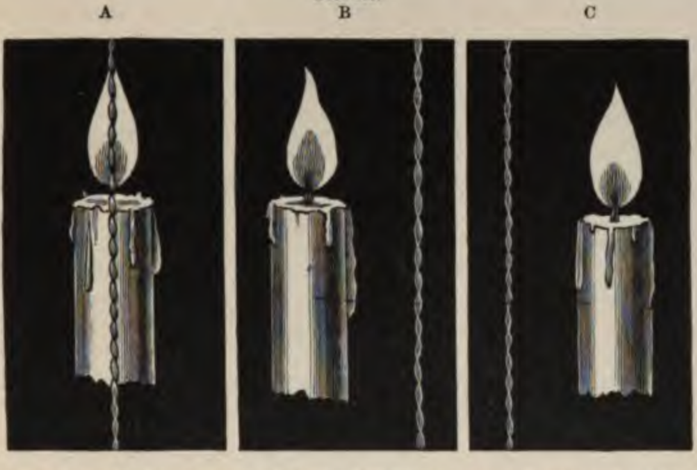
The patient is told to look at the tip of a pencil or the tip of the finger of the physician, held about three feet from his face. This object is then gradually brought nearer and nearer to him, and the eyes of the patient necessarily converge more and more as it approaches his nose. Normally the eyes will be co-ordinately converged when the object is only three and a half inches from them; but if any weakness or insufficiency of one internus is present, the eye on that side will deviate or fail to converge before this point is reached.

Again, a fine point like a pin-point is held at about eight or ten inches from the eyes and below the horizontal, and one eye is covered by a card or hand. If the eye which is separated from the object by the card deviates inward, it indicates insufficiency of the external rectus. If, on the other hand, it deviates outward, it shows insufficiency of the internal rectus. On sudden removal of the card the eye at once springs back into place for the purpose of fixing upon the object, and "in general terms each millimetre of movement deviating from the fixation-point corresponds to what is called two degrees of insufficiency, as measured by prisms" (Randall). If the internus is insufficient, and the covered eye moves in to fix in several distinct impulses, each impulse should be multiplied into the foregoing result.

A very useful, and the simplest, apparatus for testing the functional balance of the ocular muscles is the rod-test of Maddox.

A cell in which is mounted a transparent glass rod is placed in a trial frame, which is then placed in front of the eyes. If the horizontal deviation is to be determined, the physician should "seat the patient at six metres from a small flame, and place the rod horizontally before one eye, a colored glass before the other. If the line passes [vertically] through the flame, there is orthophoria (equipoise), as far as the horizontal movements of the eyes are concerned. Should the line lie to either side of the flame, as in most people it will, there is either latent convergence or latent divergence: the former if the line is the same side as the rod (homonymous diplopia), the latter if to the other side (crossed diplopia)." (Maddox.) (Fig. 193.)

FIG. 193.



Maddox's rod-test for horizontal deviation. The rod is before the right eye. A. The line passes through the flame—orthophoria. B. The line passes to the right of the flame—latent convergence, or esophoria. C. The line passes to the left of the flame—latent divergence, or exophoria. (DE SCHWEINITZ.)

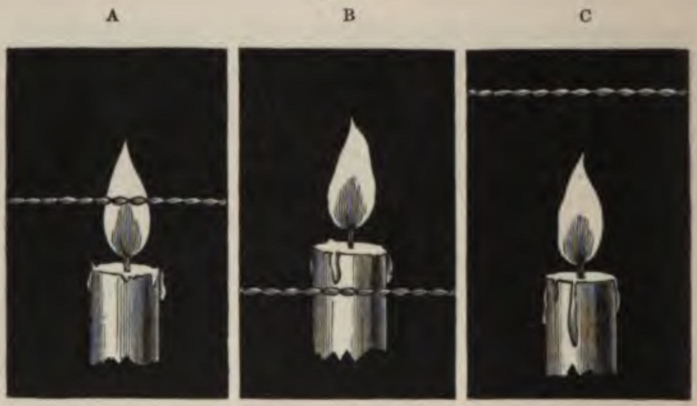
When the vertical deviation is to be estimated the rod is placed vertically in the frame. If the patient states that the horizontal line of light passes directly through the flame, the vertical balance of the eyes is normal; if, on the other hand, the line is above the flame, there is a tendency to upward deviation of the naked eye; but if the line is below the flame, there is upward deviation of the eye covered by the rod-test. (Fig. 194.)

Testing of this kind refers to the insufficiencies and not to the palsies of the ocular muscles.

The importance of being able to demonstrate these minor failures in the ocular muscles by these means lies in the fact that in this manner headaches due to muscle eye-strain may be remedied by removing their cause by properly fitted glases, or by gymnastic exercises with prisms, or in some cases by tenotomy.

Where there are marked *palsies* of the ocular muscles, there is usually some poison exercising its effects upon their nervous centres or the nerves themselves, or there is some central nervous lesion affecting the centres governing these muscles in the cortex, or there is a lesion in the nuclei or fasciculi, or, again, there may be lesions in the basal ganglia, or in the course of the fibres of the nerve between the nucleus and the eye, or in the orbit or nerve-endings.

FIG. 194.

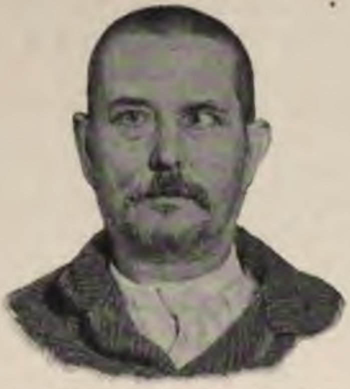


Maddox's rod-test for vertical deviation. The rod is before the right eye. A. The line passes through the flame—orthophoria. B. The line passes below the flame. The upper image belongs to the left eye—right hyperphoria. C. The line passes above the flame. The upper image belongs to the right eye—left hyperphoria. (DE SCHWEINITZ.)

The signs of paralysis of the ocular muscles consist in the following symptoms: Diplopia, which is due to the failure of the images to fall on the corresponding points in each retina. This diplopia becomes more and more marked as the object moves toward the side on which the paralyzed muscle lies. Strabismus, which may or may not be constant, usually develops when the patient endeavors to turn his eyes in the direction of the paralyzed muscle. Vertigo, which is due to the diplopia, or, if the well eye is closed, to an erroneous localization of the objects in the field of vision. Altered carriage of the head, due to the fact that the patient tries to turn his head in the direction in which he is least troubled by double images—that is, he obtains the natural fixation-point of the weak eye, and then adjusts the well eye accordingly.

If the paralysis of the ocular muscle be complete, the squint and the loss of movement of the muscle which is paralyzed will usually enable the physician to find out the paralyzed muscle; but if there be only a partial paralysis or paresis of an ocular muscle, then squint is not necessarily present, and the diagnosis of the part affected must be made by a study of the double images. This is made by placing before the patient, at a distance of from three to five yards, a candle on the same level as his eyes. One eye is covered by a piece of red glass, so that the patient can readily distinguish between the images. The lighted candle is then moved from the middle of the patient to the right and left, and the relative positions of the red and white images are noted. Then the candle is moved up and down, and the results recorded. These operations having been recorded, it is to be remembered that diplopia is most marked and sometimes only appears when the patient turns his eyes in that direction which calls into play the affected muscles, no diplopia being present if other muscles are used. Again, the image which belongs to the affected eye is projected in the direction toward which the paralyzed muscle normally turns the eye, and, finally, the distance of the double image increases when the eyes are turned in the direction of the action of the paralyzed muscle, or, in other words, that image is false and belongs to the affected eye which in the region of diplopia moves faster than the moving test-object—that is, the candle-flame.

FIG. 195.



Paralysis of left abducens in a case of hemiplegia of syphilitic origin. (DERCUM.)

If we place a candle several yards (say three to five) in front of a patient suffering from paralysis of the external rectus and at the level of his eyes, the double images of two candles will appear as in Fig. 196, if he has complete paralysis of the external rectus or internal squint of the left eye; while if the right external rectus is paralyzed, the images will appear as in Fig. 197. Further, if the object is moved to the right in the first condition, the false and the true candle separate further and further; whereas if the left externus is involved and the object is moved to the left, the same separation takes place. This condition is called homonymous diplopia, because the word homonymous indicates that the false image is seen on the same side as the eye affected.

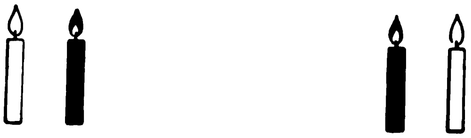


FIG. 196. FIG. 197.

The false image is in outline. (DE SCHWEINITZ.)

If, on the other hand, the false image is found to the right of the true one, as in Fig. 198, the internal rectus of the left eye is paralyzed, and causes an external squint if the paralysis is complete; and if the same muscle of the right eye is affected, the false image will be to the left side of the true one. (Fig. 199.) Further, if the object is moved to the right in the first case, the two images separate more and more widely; or if the right internus is involved, and the object is moved to the left, the same thing occurs. This is called crossed diplopia, because the image of the right eye appears on the left side, and the image of the left eye appears on the right side.

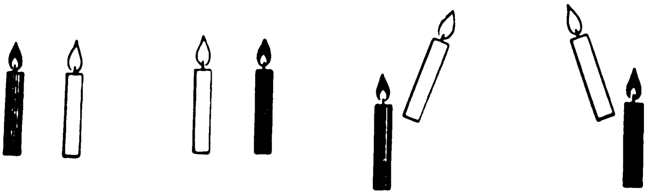


FIG. 198. FIG. 199. FIG. 200. FIG. 201.

The false image is in outline. (DE SCHWEINITZ.)

Supposing, again, that the images are seen as in Fig. 200, then the left superior rectus is involved (downward squint); while if they appear as in Fig. 201, the right superior rectus is affected. This diplopia occurs chiefly in the upper field, because, according to a rule already given, diplopia is most manifest in that portion of the field of fixation toward which the paralyzed muscle commonly rotates the eye.

If the images appear as in Fig. 202, the left inferior rectus is affected; or if as in Fig. 203, the right inferior rectus muscle. This is also crossed diplopia, chiefly in the lower field, because the inferior rectus muscle rotates the eyeball downward.

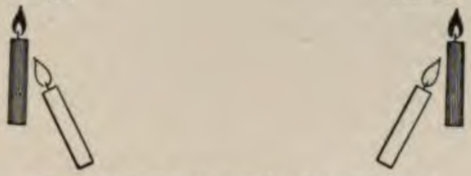


FIG. 202. FIG. 203.

The false image is in outline. (DE SCHWEINITZ.)

Again, if the images appear as in Fig. 204, the left inferior oblique muscle is paralyzed, and there will be inward and downward squint; if the right inferior oblique is affected, the images will be as in Fig. 205. There is a homonymous diplopia most marked in the upper field.

If due to paralysis of the left superior oblique, the images appear as in Fig. 206; and if as in Fig. 207, the right is affected. There is an upward and inward squint, and there is a homonymous diplopia chiefly in the lower field.

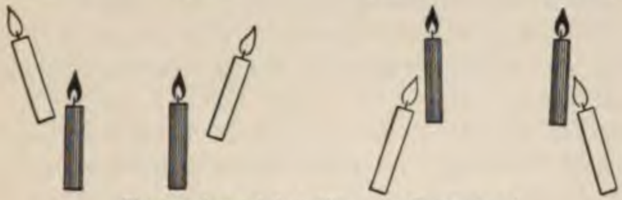


FIG. 204. FIG. 205. FIG. 206. FIG. 207.

The false image is in outline. (DE SCHWEINITZ.)

Finally, if there is divergent squint with failure of movement in all directions, except outward and slightly downward, and there are ptosis, moderate mydriasis, and paralysis of accommodation, there are oculomotor paralysis and crossed diplopia.

The following table of Hotz (*International Clinics*, vol. iii., 4th series) summarizes the facts as to the diagnosis of the conditions producing strabismus:

I. Lateral diplopia indicates paralysis of an internal or external rectus.

1. Homonymous diplopia indicates paralysis of an external rectus.

a. Images separating to the right indicate paralysis of the externus of the right eye.

b. Images separating to the left indicate paralysis of the externus of the left eye.

2. Crossed images indicate paralysis of an internus.

a. Images separating to the right indicate paralysis of the internus of the left eye.

b. Images separating to the left indicate paralysis of the internus of the right eye.

II. Vertical diplopia in the upper field indicates paralysis of the superior rectus or inferior oblique.

1. Homonymous images indicate paralysis of the inferior oblique.

a. Image of right eye higher means paralysis of the inferior oblique of the right eye.

b. Image of right eye lower means paralysis of the inferior oblique of the left eye.

2. Crossed images indicate paralysis of the superior rectus.

a. Image of right eye higher means paralysis of the superior rectus of the right eye.

b. Image of right eye lower means paralysis of the superior rectus of the left eye.

III. Vertical diplopia in the lower field indicates paralysis of the inferior rectus or superior oblique.

1. Homonymous images indicate paralysis of the superior oblique.

a. Image of the right eye higher means paralysis of the superior oblique of the left eye.

2. Crossed images indicate paralysis of the inferior rectus.

a. Image of the right eye lower means paralysis of the inferior rectus of the right eye.

b. Image of the right eye higher means paralysis of the inferior rectus of the left eye.

It is exceedingly difficult, however, always to localize exactly the affected muscle, a difficulty which is much increased when more than one is paretic, the paresis being of different degrees.

Having now considered the means of determining that the muscles are defective, we must determine the diagnostic indications presented by this examination. In other words, we must seek the cause of the paralysis or loss of power.

Paralysis of the ocular muscles may be due to a lesion in one of several places. Thus it may arise from hemorrhage, sclerosis, and softening of the cerebral cortex, in which case the other symptoms of lesions in those parts will be present as in apoplexy, disseminated sclerosis, or meningeal disease. Or it may depend upon lesions in the fasciculi between the cortex and the nuclear origin of the nerves, as in the crus. (This is rare.) Or, again, it may be due to lesions in the nuclei. If this be the case, we have developed ophthalmoplegia,[1] or paralysis of all the ocular muscles supplied by the third, fourth, and sixth nerves. This nuclear paralysis is divisible into two classes, the acute and chronic. Sometimes it is called acute and chronic nuclear palsy. The acute form is sudden in its onset, all the ocular muscles losing power. With the onset of the attack there may be fever, vomiting, and even convulsions. Such an attack results from minute hemorrhages among the nuclei, or from an acute hemorrhagic polioencephalitis in the fourth ventricle, arising from syphilis, tuberculosis, ptomaine-poisoning, alcoholic and sulphuric-acid poisoning. Such cases are usually rapidly fatal. A less fatal form follows injuries, and the effects of nicotine, lead, carbonic acid, or such diseases as diabetes, syphilis, and epidemic influenza. Sometimes acute ophthalmoplegia comes on with acute poliomyelitis or acute bulbar paralysis.

Chronic nuclear paralysis is gradual in its onset, muscle after muscle failing, and even ptosis coming on. Sometimes after a certain degree of paralysis is reached the disease comes to a standstill. The trouble may be unilateral or bilateral, and is often unsymmetrical, and it occurs after acute ophthalmoplegia, as a congenital defect producing bilateral ptosis (see chapter on Face), as an acquired disease in childhood and adult life, and in conjunction with locomotor ataxia, paretic dementia, disseminated sclerosis, progressive muscular atrophy, chronic bulbar paralysis, and in connection with paralysis of the frontalis and orbicularis palpebrarum, which are innervated by the facial nerve. The cause may be tuberculosis or syphilis, but in some cases no cause can be found.

If the cause of the paralysis of one or two muscles be basilar lesions, these may arise from hemorrhage, pachymeningitis, meningitis, both simple and tubercular, chiefly the latter; purulent meningitis, abscess as the result of middle-ear disease, and anæmia. It may also arise as the result of obliterating arteritis, particularly in syphilitics, and from tumors. The frequency with which palsy of the ocular muscles depends on syphilis is noteworthy. Alexander asserts that 75 per cent. of all cases of unilateral paralysis of the third nerve are due to syphilis, and Uhtoff states that only about 15 per cent. of the cases of cerebral syphilis escape some ocular palsy. In children sudden convergent strabismus and diplopia are often among the earliest symptoms of tubercular meningitis at the base of the brain due to pressure on the roots of the sixth nerve.

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[1] Ophthalmoplegia is here applied in its strict sense. The word is often used to signify loss of power in individual eye-muscles; and while its use in both ways is correct, it is better to confine its usage to nuclear and complete lesions.

If the cause be in the nerve-trunks themselves, the lesion will probably be cellulitis, tenonitis, hemorrhages in the orbit, or fractures of the orbit; or, again, there may be disease of the frontal sinus. If the lesion is distinctly peripheral, it may be due to rheumatism (when the external rectus is commonly affected), neurasthenia, or it may arise from uric-acid diathesis and gout. Further, such lesions may be due to influenza, diabetes, diphtheria, lead, and alcohol, or any one of the drugs which paralyze the ocular nerves.

So much for general statements as to the common and possible sites of the lesions producing paralysis of the ocular muscles. We can now go further than this, and locate the lesion more accurately from the knowledge we have gained as to the particular muscle affected and the other symptoms presented by the case.

Let us suppose that a patient suffering from paralytic internal squint or a diplopia which indicates paralysis of the external rectus, presents himself to the physician, what diagnostic significance has this symptom?[1]

In the first place, it is to be remembered that the external rectus receives its nerve-supply from the abducens, or sixth nerve (Fig. 195), which arises from the pyramidal body close to the pons. (See Plate IV. and Fig. 8.) Its deep origin is a nucleus under the floor of the fourth ventricle. The nerve pierces the dura mater on the basilar surface of the sphenoid bone (see Plate XXI.), passes through the clinoid process, enters the cavernous sinus, and, finally, enters the orbit through the sphenoidal fissure between the heads of the external rectus. If this form of squint is associated with hemiplegia of the opposite side of the body, the lesion is in the pons on the same side as the affected eye and the opposite from the hemiplegia, because the eye-fibres have crossed higher up, but the motor tracts for the limbs cross lower down.

On the other hand, if there is monoplegia and abducens palsy

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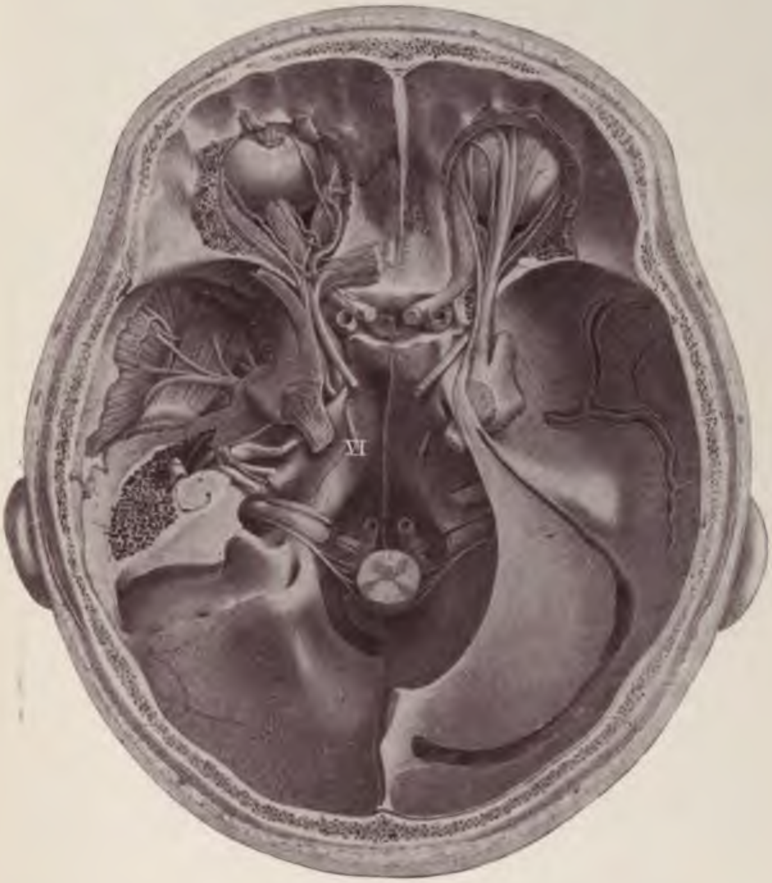
[1] This refers to paralytic and not to concomitant squint.

PLATE XXI.



Showing the Sixth or Abducens Passing through the Clinoid Process. (Modified from Arnold.)

(internal squint) on the same side of the body, the lesion is in the point of origin of the abducens and arm centre in the cortex, or, in other words, the lesion has taken place above the point where the tracts cross. Such a paralysis is, therefore, cortical.

If, again, there is complete unilateral paralysis of the abducens (internal squint) with loss of the associated action of the internus, the lesion is in the nuclei under the floor of the fourth ventricle, because the nuclei of the third and sixth cranial nerves are closely connected, so that a lesion involving the sixth nucleus weakens the nucleus of the third nerve. Complete paralysis of the externus may, therefore, be due to a nuclear lesion; for if the lesion were above the nucleus, this nucleus might obtain collateral impulses, and, therefore, the paralysis would be only partial. It may also be due to a peripheral lesion and sometimes an inflammatory process pressing upon the basilar surface of the sphenoid, and thereby involving the nerve, may cause a similar effect. Loss of power of the external rectus may also arise from neurasthenia, uric-acid diathesis, gout, and rheumatism, and in tubercular or syphilitic meningitis at the base, as already stated. It also comes on in some cases of diabetes, *la grippe*, and in chronic poisoning by lead and alcohol, or the acute poisoning of gelsemium, ptomaïne-poisoning, conium- and spigelia-poisoning.

Again, let us suppose that the internal rectus is paralyzed, causing external squint. We remember that it is supplied by the oculomotor nerve, which springs from the inner side of the crus close to the upper border of the pons (see Fig. 8 and Plates I. and IV.), very near the roots of the fourth and sixth nerves. It arises from several roots. The nerve itself pierces the dura mater below the posterior clinoid process, passes along the outer wall of the cavernous sinus, and after dividing into two branches enters the orbit through the sphenoidal fissure. (Plates I. and XXI.) The upper branch supplies the superior rectus and the levator palpebræ, and the lower one after dividing into three branches supplies the internal rectus, the inferior rectus, and the inferior oblique muscles. (Plate XIII.) The oculomotor nerve receives filaments from the cavernous plexus of the sympathetic, and from the first division of the fifth nerve. In addition to divergent squint there are, as already pointed out in the last few pages, in oculomotor paralysis, as additional symptoms, ptosis, mydriasis, and paralysis of accommodation. The lesion producing unilateral ptosis may be found in the cerebral cortex on the opposite side from the affected eye in the angular gyrus just below the interparietal fissure. Again, tubercular or other degenerative disease of the corpora quadrigemina may cause double ptosis.

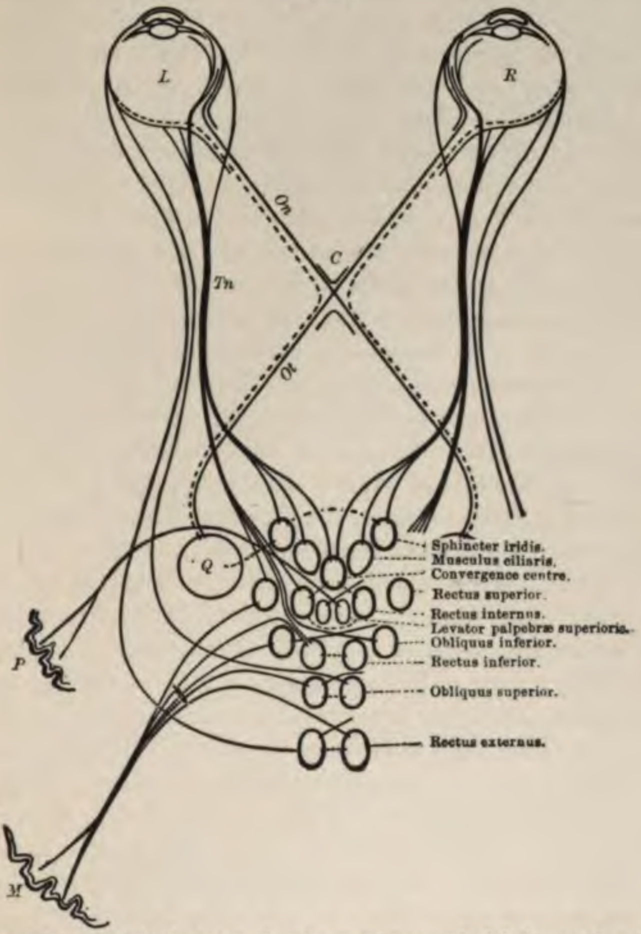
If the patient has ptosis with preservation of the function of the intraocular muscles (that is, partial oculomotor paralysis), with hemiplegia of the opposite side of the body, the lesion, according to Mauthner, probably affects the fascicular fibres—that is, those between the cortex and the nuclei. There may be associated with this form of oculomotor paralysis loss of power in the hypoglossal and facial nerves. On the other hand, if the oculomotor paralysis is complete, the lesion is almost certainly at the base of the brain, and this diagnosis becomes practically certain if there is associated with it paralysis of other cranial nerves. Paralysis of the oculomotor nerve on one side with hemiplegia on the opposite side of the body is not positively a crus or fascicular lesion unless the paralysis occurs simultaneously. (Hughlings Jackson.)

If, however, there be double oculomotor paralysis, the lesion is bilateral and probably due to a lesion at the base, as meningitis or arteritis (see Plate I.), or to an inflammatory exudate involving both nuclei; or, again, to diphtheritic poison, or the lesions of tabes dorsalis.

If that very rare form of ocular muscle paralysis, namely, isolated palsy of the fourth, or trochlear, nerve, is present, we will probably find that the paralysis is due to a lesion at the base of the brain, due to meningitis, or the pressure of a growth.

Supposing, however, that a patient presents himself with swelling of the eyelids, exophthalmos, a contracted, followed by a dilated pupil, anæsthesia of the skin of the upper eyelid and of the temple, or the area supplied by the first division (ophthalmic) of the fifth nerve, and ophthalmoplegia — that is, paralysis of the extrinsic ocular muscles on one side—where will be the lesion productive of this train of interesting symptoms? It will be seen at once that such a condition is the result of paralysis of the oculomotor (third), pathetic (fourth), and abducens (sixth) nerves, and that, as in all probability only one lesion has produced these symptoms, it must exist at some point where all these nerve-fibres are so closely approximated that they are readily involved together. It will be recalled that the course of these nerves is as follows: the oculomotor nerve, having arisen from the nucleus in the corpora quadrigemina, pierces the dura mater below the posterior clinoid process, passes along the outer wall of the cavernous sinus, and there divides into two branches.

FIG. 208.



Scheme of the nuclei of the nerves of ocular movement and of their central and peripheral tracts: *R*, right eye; *L*, left eye; *C*, chiasm; *On*, optic nerve; *Ot*, optic tract; *Q*, pregeminum (anterior quadrigeminal body); *P*, cortical centre for the movement of elevation of the upper eyelid; *M*, cortical centre for ocular movements; *Tn*, course of all ocular nerves in the cavernous sinus. The names of the different nuclei are printed on the diagram, and the nerve-tracts going from these nuclei can be readily traced to where they converge in their course in the cavernous sinus and where they diverge to pass the various muscles of the eye. The dotted lines represent associating and commissural tracts, showing how the fibres of the third, fourth, and sixth nerves come together in one bundle in the cavernous sinus. (From Mill's *Nervous Diseases*.)

The pathetic nerve passes near the clinoid process along the outer wall of the cavernous sinus, and with the oculomotor nerve enters the orbit through the sphenoidal fissure. The sixth nerve pierces the dura mater on the basilar surface of the sphenoidal bone, passes through the clinoid process, and enters the cavernous sinus, finally reaching the orbit through the sphenoidal fissure. It is thus seen that a lesion at the sphenoid fissure and pressure in the cavernous sinus would cause all the symptoms described above. (Fig. 208 and Plates I. and XXI.) This occurs in cases of thrombosis of the cavernous sinus. Where there is an arterio-venous aneurism of this sinus there will be pulsating exophthalmos. Injury or inflammation, if in this area, may also produce a series of symptoms.

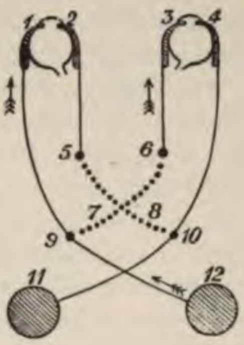
The significance of conjugate lateral paralysis producing a deviation of both eyes to the right or left, as the case may be, is that some lesion exists in the cerebral cortex, the corona radiata, or the internal capsule, or in the pons before the fibres have crossed. The lesion, if in the cortex, however, does not have to be localized in one spot, for any source of irritation in the cortex may apparently cause conjugate deviation. If the lesion is the result of an apoplexy, the eyes are turned toward the side opposite to the paralysis (Prevost's symptom)—that is, the "patient looks at his lesion." The reason that a unilateral lesion can cause a bilateral deviation is that the lateral movements of the eye are governed by an impulse which passes down from the cortex to the sixth-nerve nucleus, and thence across the posterior longitudinal fasciculus to the opposite side, where it passes to the nucleus of the third nerve. As conjugate lateral deviation is caused by contraction of the internal rectus on one side (third nerve) and the external rectus on the other (sixth nerve), the mechanism of the deviation is clear. Thus, if the lesion be a distinctive one on the left side of the brain, causing right hemiplegia, the eyes will be turned to the left by the action of the unaffected left external rectus and the right internal rectus; while if the lesion be on the right side of the brain, the reverse will occur. If, however, the lesion be irritative (as a tumor), this deviation is reversed, because in this case the centres are irritated and cause spasm of the muscles receiving their nerve supply from the affected side of the cerebrum. In other words, the eyes are turned toward the side of the body which is convulsed.

In the first instance the eyes are turned away from the affected side because the muscles of the eyes on that side are also paralyzed, and the eyes are, therefore, turned by the muscles which remain intact. In the second instance the eyes are turned toward the convulsed side because the internal and external rectus are spasmodically contracted and so overcome the healthy muscles.

We find, however, that if the lesion be in the pons rather than in the cortex, these conditions are reversed, for now a destructive lesion causes the eyes to be turned to the paralyzed side, and an irritative lesion directs them away from the paralyzed side.

This is best explained by the following diagram and description from Swanzy's well-known book. (Fig. 209.)

FIG. 209.



1. Left ext. rectus. 2. Left int. rectus. 3. Right int. rectus. 4. Right ext. rectus. 5. Nucleus left third nerve. 6. Nucleus right third nerve. 7 and 8. Post. longitudinal bands from sixth nerve to opposite third nerve. 9. Nucleus left sixth nerve. 10. Nucleus right sixth nerve. 11 and 12. Left and right cortical centres. An impulse starting from 12 would travel down to 9, and produce an associated movement of the eyes to the left.

A destructive lesion at 12, the right cortical centre, involving also motor centres of the body, would cause left hemiplegia; and, since the external rectus of the left eye and internal rectus of the right eye would be paralyzed, the antagonists would turn the eyes to the right—*i. e.*, away from the paralyzed side. A destructive lesion of the right side of the pons, also producing left hemiplegia, if it involves the sixth nucleus, will produce paralysis of the external rectus of the right eye and of the internal rectus of the left eye, and then the antagonists would turn the eyes to the left—*i. e.*, toward the paralyzed side. It is easy to see how irritative lesions would produce exactly the opposite effects.

Squint which is due to hysteria is always caused by spasmodic contraction of the eye-muscle and is never due to paralysis, as it often is in organic disease. Very often there is a spasm of the eyelid or eyebrow associated with it. The administration of a relaxant, such as chloroform, will at once overcome such a squint.

Nystagmus, or the rapid oscillation of the eyes from side to side or in a vertical or rotary movement, is usually bilateral.[1] When not congenital it is a frequent symptom of disseminated sclerosis, Friedreich's ataxia, and advanced locomotor ataxia, and while it does not localize the lesion it indicates very positively that one is present and that the case is not one of hysteria or functional disease. Nystagmus occurring in children is very often associated with imperfect vision of great degree or with blindness as a result of opacity of the cornea, congenital cataract, or atrophy of the nerve. In other instances, however, it arises from growths in the cerebellum or pons, and it is sometimes seen in hydrocephalus and very rarely in acute meningitis and in epilepsy. Very rarely lateral nystagmus is seen in children who seem otherwise normal, and it then possesses no particular diagnostic importance.

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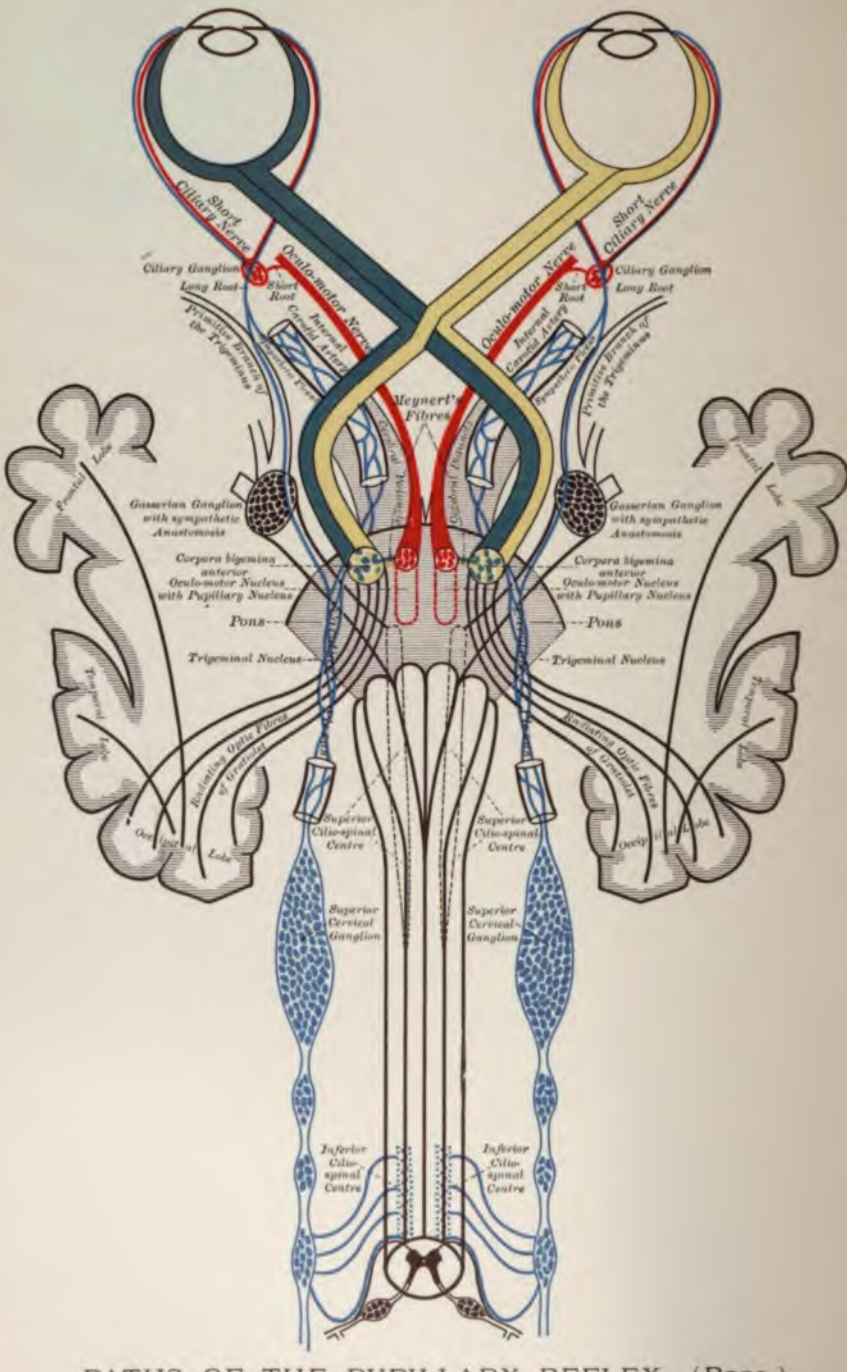
[1] The physician should remember that some occupations, such as mining, produce in some persons nystagmus without the presence of the disease about to be named.

Paralysis or Disorder of the Intraocular Muscles. Having discussed the diagnostic indications of changes in the functions of the extraocular muscles, we next proceed to a consideration of these facts in connection with the intraocular muscles. These consist, it will be remembered, in the muscular fibres of the iris, circular and radiating, and the ciliary muscle. The nerve-supply of the iris consists in fibres from the oculomotor or third nerve, the upper or ophthalmic division of the fifth, and the sympathetic. It will be remembered that in the posterior part of the orbit there is situated a ganglion called the ciliary or ophthalmic ganglion. By its short or motor root this ganglion is connected with the third nerve, by its sympathetic root with the cavernous sympathetic plexus and the cervical sympathetic plexus, while by its long or sensory root it is connected with the nasal branch of the ophthalmic or upper branch of the fifth nerve. From this ganglion extend forward two sets of nerves, one short (the short ciliary nerve), which supplies the iris and the ciliary muscle, and one set long (long ciliary nerves), which also go to the iris. The filaments which go to the ganglion by means of its short or motor root (from the oculomotor nerve) pass forward to the circular fibres of the iris, while those which have arisen in the sympathetic plexus pass forward to the radiating fibres. These last fibres are in part derived from the cervical sympathetic ganglion, run through the carotid plexus, and are controlled to some extent by the cilio-spinal centre of Bunge in the spinal cord at about the seventh cervical or first dorsal vertebra.

The ciliary muscle is supplied by the fibres of the short ciliary nerves, which have arisen in the floor of the third ventricle and which is connected with the nucleus of the third nerve.

PLATE XXII.


Short Ciliary Nerve
Oculo-motor Nerve
Ciliary Ganglion
Long Root
Short Root
Internal Carotid Artery
Primitive Branch of the Trigeminus
Sympathetic Plexus
Meynert's Fibres
Frontal Lobe
Gasserian Ganglion with sympathetic Anastomosis
Corpora bigemina anterior
Oculo-motor Nucleus with Pupillary Nucleus
Pons
Trigeminal Nucleus
Temporal Lobe
Radiating Optic Fibres of Gratiolet
Occipital Lobe
Superior Cilio-spinal Centre
Superior Cervical Ganglion
Inferior Cilio-spinal Centre
Short Ciliary Nerve
Oculo-motor Nerve
Ciliary Ganglion
Long Root
Short Root
Internal Carotid Artery
Primitive Branch of the Trigeminus
Frontal Lobe
Gasserian Ganglion with sympathetic Anastomosis
Corpora bigemina anterior
Oculo-motor Nucleus with Pupillary Nucleus
Pons
Trigeminal Nucleus
Temporal Lobe
Radiating Optic Fibres of Gratiolet
Occipital Lobe
Superior Cilio-spinal Centre
Superior Cervical Ganglion
Inferior Cilio-spinal Centre

PATHS OF THE PUPILLARY REFLEX. (Baas.)

The green line connecting the Corpora Bigemina Anterior and the Oculo-motor Nucleus represents Meynert's Fibres and the site of a Lesion, causing Unilateral Reflex Iridoplegia is shown in red lines outlining the Oculo-motor Nucleus.

Testing the Pupil. The normal pupil is about four millimetres in diameter, but this varies according to the degree of light to which the patient is exposed. It ought always to be measured by a millimetre measure, which gives its approximate diameter.

The pupil to be tested must be free from any abnormal conditions produced by new or old inflammation of the iris, and the light used should not be excessively bright, but about that usual to the eye.

The patient is to be placed facing the light and told to look at some distant object. The hands of the physician are then placed one over each eye, the patient being told to keep his eyes open and to endeavor to see the object seen before the eyes were covered. One hand is now quickly removed from one eye and the pupil observed. This observation must be acute or the pupillary contraction will occur before it is seen. This reflex is due to the fact that we have an irritation of the optic nerve by light, and this sends a reflex wave to the centres governing the pupil and causes it to contract. Not only does the uncovered pupil react in this manner, but the covered one does the same thing. The first is called a direct reflex, the second is called the indirect or consensual reflex. The exact pathway of this reflex is unknown, but we know that the light-impulse passes along the optic nerve, and arriving at its semidecussation in the chiasm, passes along each of the tracts to the corpora quadrigemina, and thence by the communicating fibres (Meynert's fibres, see Plate XXII.) between these bodies and the oculomotor centres to the centre for the sphincter pupillæ or circular muscle-centre, and from there to the ciliary ganglion, the ciliary nerves, and the muscles of the iris.

Not only does the pupil change its size by reason of the ordinary light-reflex, but it also contracts or dilates in association with the other muscles governing accommodation and convergence, namely, the ciliary muscle and internal recti. This is the associated reaction of the pupils, and is tested by causing the patient to direct his eyes to a near object—for example, the point of a pencil. If the sight is intact, contraction of the pupil will occur.

The pupil-dilating centre is in the medulla and is very sensitive to reflex irritation.

Supposing that the pupillary movement is abnormal, we should recollect before studying the case further what the causes of its perversion may be. Thus, its size is altered by drugs, by local disease of the iris, by spinal disease and disease of the sympathetic, by localized cerebral lesions, by abeyance of the cerebral functions, and by irritation of the brain. Let us suppose, however, that on testing the ocular reflexes in the manner already described we find that the pupil of one eye when uncovered does not contract, and immediately does so as soon as the other eye is uncovered, what is the indication? It indicates that there is disease of the optic nerve of that eye which does not convey the impulse of light from the retina; whereas if it contracts when the other eye is uncovered, it shows that the rest of the mechanism involved in the reflex is intact. Accommodative reaction of the pupil is intact also. Again, supposing that the irides fail to react to light, but do to accommodation and convergence, we have the "Argyll-Robertson pupil," so called, which indicates that a lesion exists in the communicating fibres (Meynert's fibres, see Plate XXII.) which convey the impulses from the corpora quadrigemina to the oculomotor nuclei. The fact, however, that unilateral reflex iridoplegia may occur indicates that the lesion does not always involve Meynert's fibres, but rather that the lesion is situated in the anterior part of the oculomotor nuclei. In other words, unilateral reflex iridoplegia, if present, shows a lesion in the oculomotor nuclei or its efferent branches.

This condition is seen in locomotor ataxia, general paralysis of the insane, sometimes in cerebral syphilis, and as the result of poisoning by the bisulphide of carbon. Recently Grube has reported three cases in which the Argyll-Robertson pupil developed in the course of diabetes mellitus. Marinesco has reported an instance of Argyll-Robertson pupil in a patient suffering from amyotrophic lateral sclerosis. It is worthy of note, however, that late in all these affections the reaction to accommodation may also be lost. Rarely the reverse of the Argyll-Robertson pupil occurs as the result of a lesion in the second and third parts of the oculomotor nucleus. If the eyes fail to react to light and to accommodation, there is probably blindness due to optic-nerve disease.

If on throwing light into the right eye there is no reaction of the pupil of that eye, and on throwing it into the left eye there is still no reaction in the pupil of the right eye, there must be a lesion of the nucleus of the right oculomotor nerve or palsy of the conducting fibres of each optic nerve.

Sachs asserts that immobility of the pupil is very characteristic of syphilitic cerebro-spinal disease, and if the diagnosis lies between multiple sclerosis on the one hand, and cerebro-spinal syphilis on the

the other, the discovery of immobility of one or both pupils should decide in favor of its being a syphilitic case. He also asserts that persistent pupillary immobility in a case of hemiplegia indicates a syphilitic endarteritis. It is important in this connection to remember that the pupillary changes due to syphilis often suddenly improve, while those due to sclerosis are absolutely permanent.

Contraction of the pupil occurs in a large number of conditions, and yet possesses considerable diagnostic significance. Thus it is generally found in the early stages of all acute inflammatory processes in the brain or its membranes. In cerebral hemorrhage it is usually contracted at first, thus serving to separate acute paralysis due to hemorrhage from that due to embolism, for in the latter Berthold states that the pupil is unaltered. In the early stages of intracranial tumors which irritate the third-nerve nucleus it is also contracted. Finally, we find myosis as a result of chronic tobacco-poisoning, from irritation of the pupil-contracting centre by nicotine, at the beginning of an attack of hysteria or epilepsy, and in watch-makers and jewellers. Such forms of myosis are called "irritative myosis."

Paralytic myosis is met with in lesions above the dorsal vertebræ of a chronic type, as a rule. Its most interesting form is that seen in locomotor ataxia, when the disease has involved the cilio-spinal centre. When the lesion also attacks Meynert's fibres we meet with the Argyll-Robertson pupil.

Of course, pupillary contraction may result from the action of a myotic drug, as eserine or pilocarpine.

Dilatation of the pupil may also be due to irritation or paralysis. Thus, irritation of the pupil-dilating centre may cause mydriasis, and this is met with in congestion of the cervical spinal cord and in spinal meningitis, as the result of tumors in the cervical cord, in spinal irritation, in the anæmia of convalescence, as an early sign of tabes dorsalis, and in acute mania. Certain cases of acute croupous pneumonia present dilatation of both pupils, the dilatation being most marked on the affected side probably by irritation of the sympathetic fibres by pressure of the consolidated lung. Sometimes in progressive paralysis of the insane there may be irritative mydriasis in one eye and myosis in the other. Von Graefe asserted that alternating unilateral mydriasis is an early sign of mental derangement.

The states in which we find paralytic mydriasis are in the later stages of progressive paralysis, in lesions at the base affecting the oculomotor centre, late in thrombosis of the cavernous sinus, in orbital disease which causes pressure on the ciliary nerves, in glaucoma, and in intracranial growths of considerable size. Not only may paralytic mydriasis be due to an oculomotor lesion, but as the result of some blocking of the pathway from the retina to the centre.[1]

Under the name of "hemiopic pupillary inaction" or "Wernicke's pupil," we sometimes, though rarely, meet with a condition associated with hemianopsia, or blindness in one-half of the eye, which is demonstrated in the following manner: the patient is seated in a dark room and one eye is covered. The other eye is now illuminated by just sufficient light from a flat mirror to enable the physician to see the eye. By means of the concave mirror of an ophthalmoscope the physician now directs into the uncovered eye a bright beam of light, taking care that it falls upon one side of the retina, or, in other words, enters the eye obliquely and strikes on the side of the retina which is blind. If when the light falls on the blind side of the retina there is no pupillary reaction, it is considered that the lesion exists in the arc between the optic chiasm and the corpora quadrigemina; but if there is a pupillary reaction, the lesion must be further back in the visual centres, back of the reflex arc. When the lesion is found back of the reflex arc it may indicate a lesion of the optic tract, the posterior segment of the thalamus, the posterior part of the chiasm, or rarely it may be caused by a lesion of the optic nerve if the hemianopsia be monocular, which is rarely the case.

Finally, a rhythmical contraction and dilatation of the pupil, called "*hippus*," is seen in health for a moment on sudden exposure to light; but when constant is a sign of disseminated sclerosis, epilepsy, or the early stages of acute meningitis. It is sometimes seen in hysteria.

The presence of a recurrent, unequal dilatation of the pupils of a transitory character is said by Rampoldi to be an early and almost constant sign of pulmonary tuberculosis. He believes that this is due to a reflex irritation of the nerves governing the pupil through the sympathetic system. Probably in these cases enlarged glands in the chest are the cause of the pupillary phenomenon, just as an aneurism may be. Destree claims that 97 per cent. of his cases of phthisis present this pupillary symptom.

Knies points out that pupillary contraction and dilatation take place in association with Cheyne-Stokes breathing. Dilatation usually exists with the inspiratory movements, and myosis occurs during the interval of apnœa.

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[1] For a useful summary of these facts and for references, see "Diseases of the Eye," by Swanzy, sixth ed.

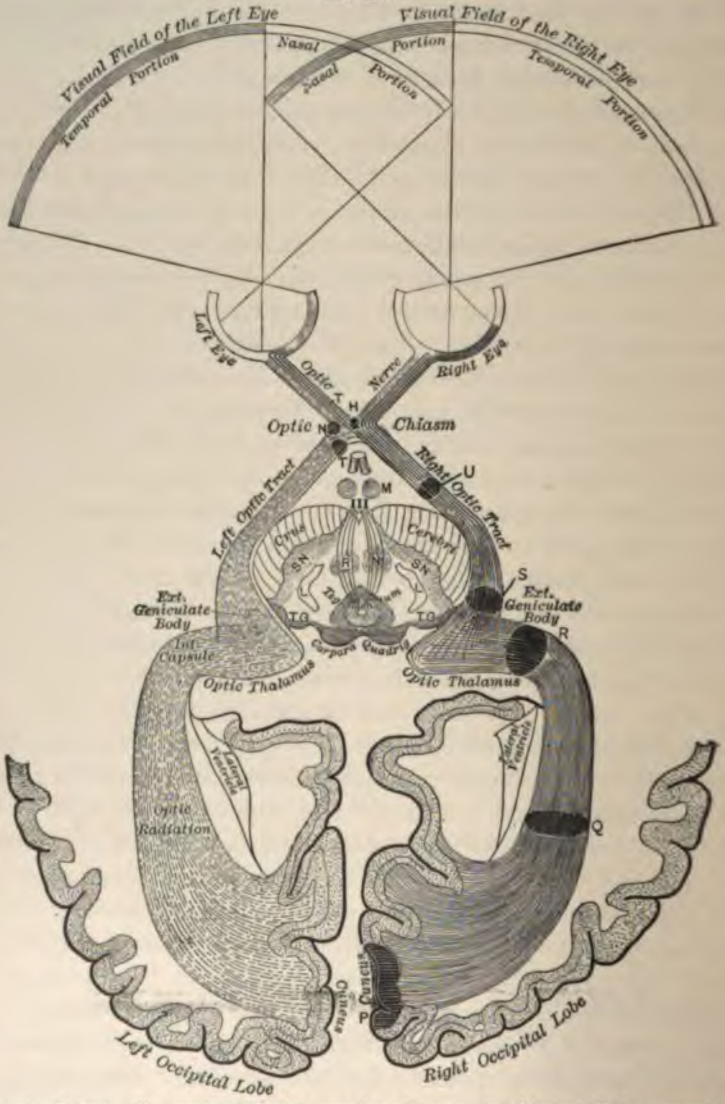
Changes in the Acuity of Vision. Having discussed the diagnostic value of alterations from the normal in the function of the extra- and intraocular muscles of the eye, we can proceed to a consideration of the value of changes in the acuity of vision. The questions of the acuity of vision in relation to errors in the refractive media of the eye will not, of course, be included in this book.

Failure of vision in part or *in toto* depends upon a lesion which destroys the peripheral ocular sense-organ (the eye), the optic nerves, the optic tracts, or the receptive and perceptive centres of sight. It also is dependent upon bilateral lesions in the crystalline lens, as in cataract, or in the cornea, as in severe keratitis.

Before we discuss these various causes of blindness it is necessary that we recall the nervous anatomy of the organs of sight. These nerve-fibres starting with the rods and cones of the retina and the fibres from the macula pass back along the optic nerve until they come to what is known as the chiasm, where the various fibres from the eye decussate, in that the fibres from the inner half of each eye cross to the opposite side, whereas those of the outer half of each eye pass to the same side, as is shown in Fig. 210. After the optic tracts have been formed by this (partial) decussation each one winds around the corresponding crus cerebri, and terminates in two roots upon the corpora geniculata externa and interna and upon the posterior part of the optic thalamus. The pupillary fibres also branch here to the corpora quadrigemina. These parts are known as the primary optic centres. After leaving them the fibres pass backward into the posterior part of the posterior limb of the internal capsule and thence to the cortex, rise in a fan-shape, pass outside the tip of the lateral ventricle, and reach the secondary or true optical centre in the lower part of the median aspect of the occipital lobe. (See Fig. 210.)

HEMIANOPSIA. As lesions of the nervous centres frequently produce partial or complete blindness, it is of importance, first, that the presence of partial blindness should be discovered, and, second, that the lesion causing it should be located. Aside from general failure of vision due to changes in the retina or optic nerve we have in many cases of nervous disease a condition called hemianopsia or partial or complete blindness of one-half of the retina. Usually hemianopsia is bilateral—that is, in both eyes; and it is usually homonymous—that is, on the same side of each eye; or, in other words, if it is in the outer half of the left eye, it will be in

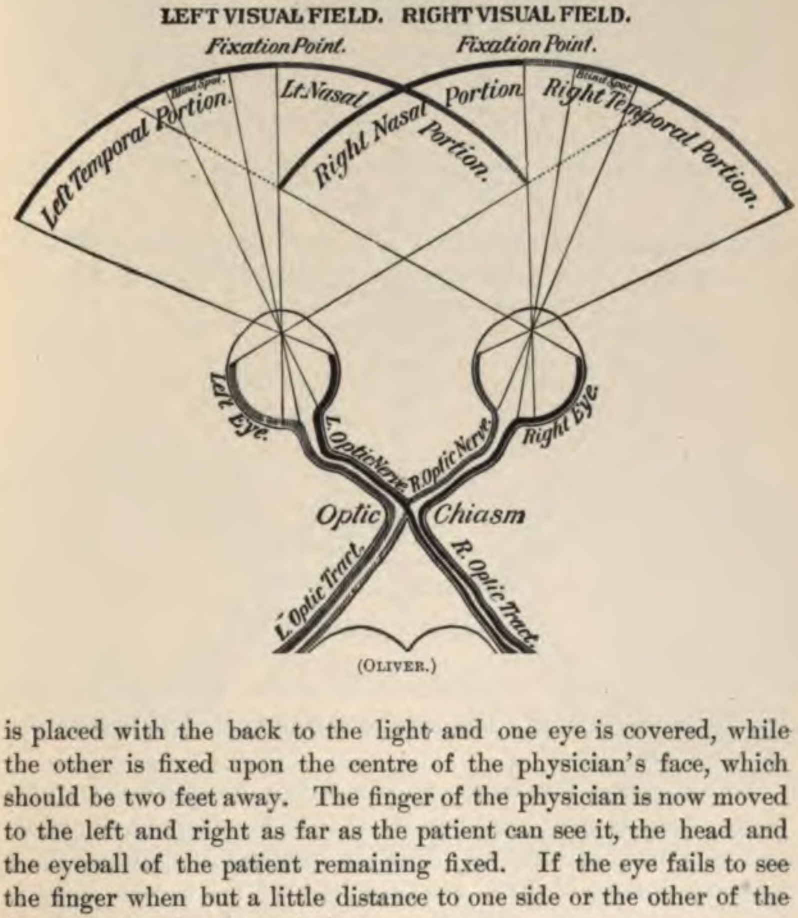
FIG. 210.



The visual tract. The result of a lesion anywhere between the chiasm and the cuneus is to produce homonymous hemianopsia. H. Lesion at chiasm causing bilateral temporal hemianopsia. N. Lesion at chiasm causing unilateral nasal hemianopsia. T. Lesion at chiasm causing unilateral temporal hemianopsia. SN. Substantia nigra of crus. L. Lemniscus in crus. RN. Red nucleus. III. Third nerves. P, Q, R, S, U. Lesions in the occipital lobe and in front of it, producing left homonymous lateral hemianopsia.

the inner half of the right eye. If this is the case, it is called left bilateral homonymous hemianopsia. If, on the other hand, the outer half of each eye is blind, this is called bitemporal hemianopsia; if the blindness is found in the nasal side of both eyes, it is called binasal hemianopsia. It must be remembered, however, that the apparent blindness of the outer side of the eye is really due to disease of the fibres supplying the opposite side of the retina, as is shown in Fig. 211. The presence of hemianopsia in any form is determined by the following method of examination: the patient is placed with the back to the light and one eye is covered, while the other is fixed upon the centre of the physician's face, which should be two feet away. The finger of the physician is now moved to the left and right as far as the patient can see it, the head and the eyeball of the patient remaining fixed. If the eye fails to see the finger when but a little distance to one side or the other of the fixation-point, hemianopsia is present.

Fig. 211.



(Oliver.)

We measure the field of vision more accurately by means of what is known as a perimeter, which is a semicircular metal band which revolves upon its middle point, being capable therefore of describing a hemisphere in space. This arc is divided into degrees marked on it from 0° to 90° and at the centre of it is placed the eye which is to be examined, which eye finds its fixation-point in the centre of the semicircle. A small piece of white paper is now moved along the metal arc on its inner surface, from the extremity and toward the centre, until it comes into view, when the physician notes the

FIG. 212.

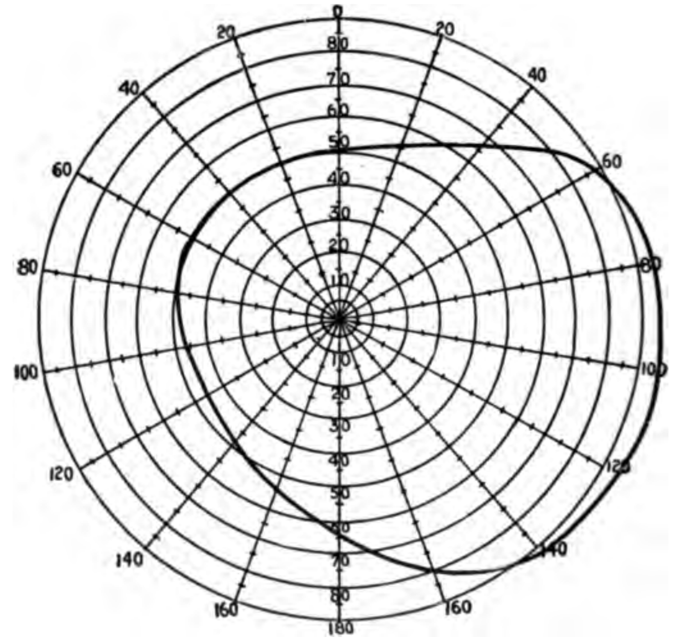


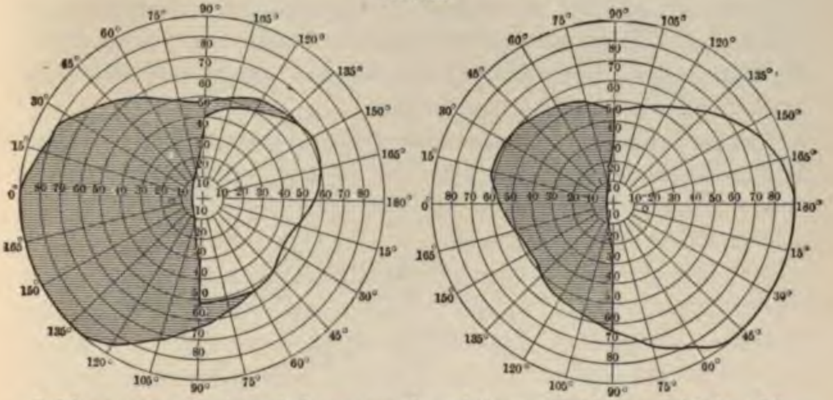
Chart of F. V. of right eye.

number of degrees at which the object is seen and marks it on a chart. (See Fig. 212.) The area of the normal field is well seen in this figure.

Let us suppose that on using the tests just described we find left lateral homonymous hemianopsia—that is, blindness in the visual field, as shown in Fig. 213. This signifies that the patient has a lesion somewhere in the visual tract back of the chiasm, either in the cuneus, in the occipital lobe, in the optic radiations, in the internal capsule, in the primary optic centres, or in the optic tract. Fig.

209 shows the sites of these lesions and why they cause left homonymous hemianopsia. Supposing, on the other hand, that instead of

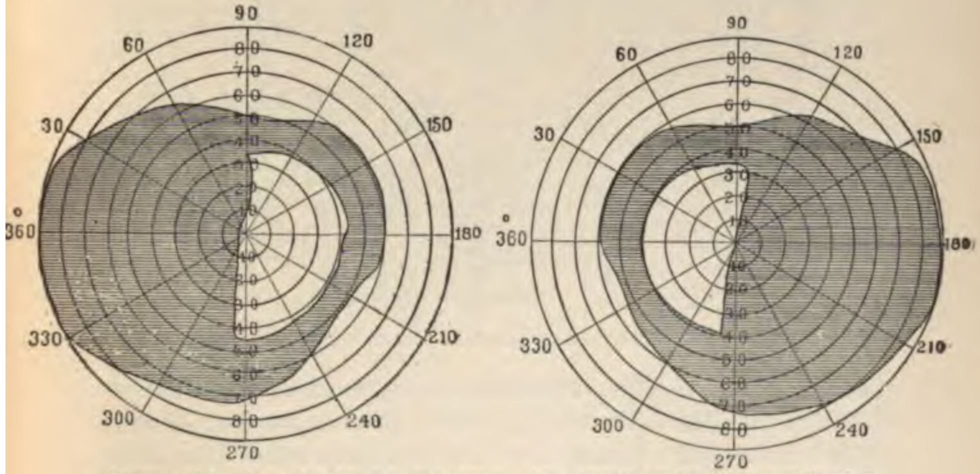
FIG. 213.



Left homonymous hemianopsia from a case of gunshot-wound, with suspected lesion of the right cuneus. (DE SCHWEINITZ.)

left homonymous hemianopsia we find bitemporal hemianopsia (Fig. 214), this indicates that the patient has a lesion of the optic tracts in

FIG. 214.



Bitemporal hemianopsia from a case of acromegaly originally under the care of Dr. H. C. WOOD, and later studied by Dr. PACKARD. Eyes examined in 1885 by DR. G. E. DE SCHWEINITZ, and above fields found. (DE SCHWEINITZ.)

the crossing fibres in the middle of the chiasm (see "H" in Fig. 210); or if binasal hemianopsia, that he has a lesion on both sides of the chiasm or one on the outer side of each optic nerve. This is a very rare lesion.

Hemianopsia of the homonymous form is very rarely found in hysteria, generally in association with hysterical hemianæsthesia, in which condition the conjunctiva is usually anæsthetic, thereby differing from the condition of the conjunctiva of persons suffering from hemianæsthesia of an organic origin.

In some cases in place of hemianopsia we have simply an alteration in the visual fields for color. It will be remembered that the boundaries of the power of the clear perception of colors are not identical with the boundary for white light, nor are they identical with one another. Passing from the periphery toward the centre of the visual field in ordinary daylight we find that blue is the color first seen, its boundary being almost as great as that of white. After blue come yellow, orange, red, and finally green. The blue, red, and green being the most important colors, their boundaries are shown in Fig. 215. These fields are determined by means of small

FIG. 215.

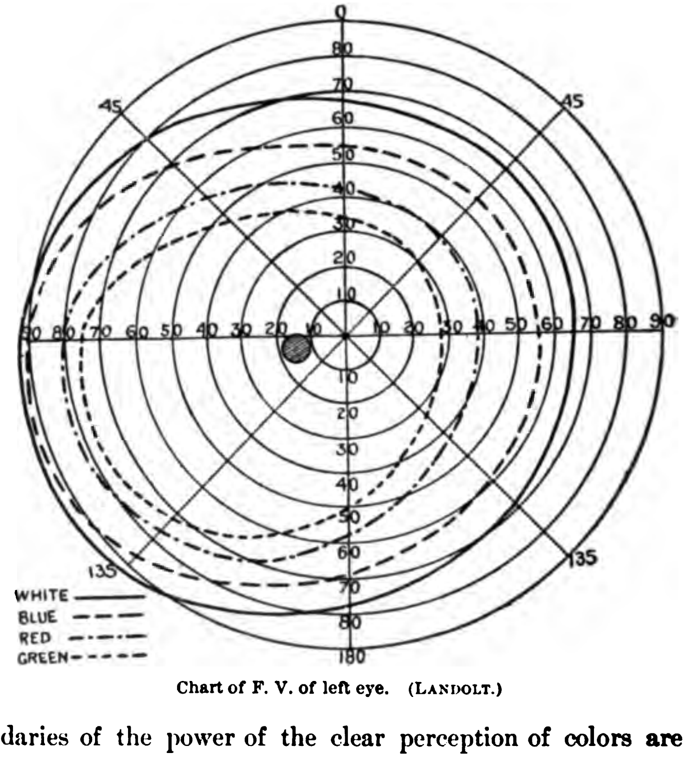


Chart of F. V. of left eye. (LANDOLT.)

pieces of colored paper passed around the perimeter in the manner already described.

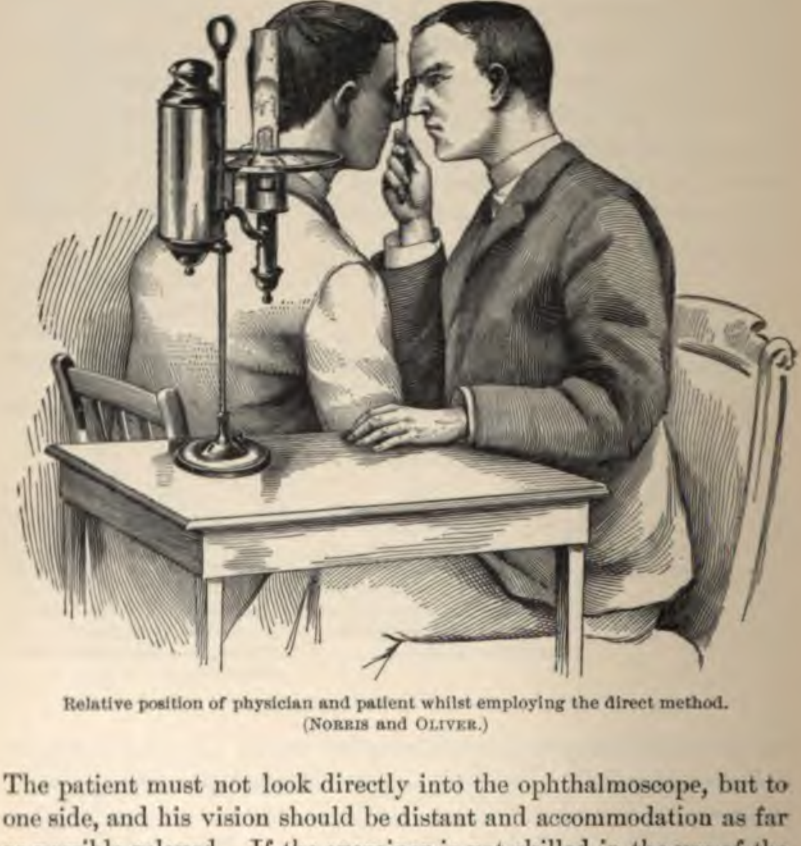
The alteration of the visual field for colors is called, if so changed, homonymous hemidyschromatopsia, and the lesion producing it is situated in the cortex of the occipital lobe; while if the colors are indistinguishable, it is called hemiachromatopsia. This site of the lesion has recently been denied. The transposition of the visual fields for color is usually a symptom of hysteria, and, as a rule, the red field takes the place of the blue, and *vice versa*. The fields for all the colors are also markedly narrowed in hysteria. This transposition, rather than loss of color-sense, helps us sometimes to a distinction between the ocular symptoms of hysteria and those of true tabes dorsalis, a distinction which is of great importance, yet one which is often exceedingly difficult, save for these and two other symptoms, namely, that in hysteria the knee-jerks are usually preserved and the Argyll-Robertson pupil is not seen. The following table from Charcot's lectures for 1888–'89 summarizes these differential points :

	Tabes.	Hysteria.
Motor apparatus of the eye.	Paralysis from lesion of a motor nerve of the eye (bulbar or peripheral) ; consequent diplopia.	1. Sometimes associated paralysis. 2. Blepharospasm. 3. Monocular diplopia ; micropsia and macropsia.
Pupillary disturbances.	Argyll-Robertson pupil.	
Optic disk.	Atrophy.	
Symptoms due to affections of the optic nerve or visual centres.	1. Irregular concentric contraction of the visual fields. 2. *Tabetic* achromatopsia or dyschromatopsia, affecting first green and red, yellow and blue being preserved to the last. 3. Progressive blindness.	1. Regular concentric contraction of the visual fields. 2. Dyschromatopsia from simple contraction of the visual fields for colors. Frequently perception of red alone persists. 3. Transitory amblyopia or amaurosis.

The Optic Nerve and the Ophthalmoscope. There still remain to be considered the diagnostic indications afforded us by the optic nerve. Before taking up this subject mention must be made of the manner of using the ophthalmoscope.

The patient is to be seated in a darkened room, and by his side, at the level of the eye to be examined and far enough back of him for his face to be in shadow, should be placed a lamp, or, if gas can be had, an Argand burner. The physician now seats himself, if the right eye is to be observed, at the right side of his patient, and takes a chair slightly higher than that of the patient. The ophthalmoscope is now taken in the right hand and held in such a position that the concavity of the physician's brow fits over the convexity of the instrument. The eye of the physician is so placed that he can readily see through the aperture in the centre of the ophthalmoscope, and by means of the concave mirror on the face of the instrument he reflects the light into the eye through the pupil.

FIG. 216.



Relative position of physician and patient whilst employing the direct method. (NORRIS and OLIVER.)

The patient must not look directly into the ophthalmoscope, but to one side, and his vision should be distant and accommodation as far as possible relaxed. If the examiner is not skilled in the use of the ophthalmoscope and the result of the examination is of great importance in the diagnosis of the case, it is justifiable to use homatropine to dilate the pupil and prevent the alterations of accommodation by paralyzing this function. The ophthalmoscope and the head of the physician are now approached as closely as possible to the eye of the patient, the angle of the two heads being as nearly as possible identical, as shown in Fig. 216. If the light be now directed slightly toward the nasal side of the eye, the optic nerve will be seen, or in its stead a retinal bloodvessel will be seen across the field of vision, and this should be traced along its course to its origin in the papilla. If the patient or the physician is short-sighted (myopic), the ophthalmoscope must be adjusted to correct this error by placing over the aperture a concave lens; but if ordinary degrees of far-sightedness (hypermetropia) are present, the use of a convex lens is not necessary, because the accommodation of the eye makes up for the error in refraction. If the hypermetropia is so great that accommodation cannot overcome it, then a convex lens must be used. The view of the eye which is obtained ordinarily by a beginner is clouded, not because of myopia or hypermetropia, but because the physician has not as yet learned to relax his accommodation in making the examination. A concave glass usually remedies this.

In health the optic nerve appears as a nearly round or slightly oval disk, situated somewhat to the nasal side of the eye, and varying in color from grayish-pink to red, the centre being whiter and the nasal half the darkest part. Around the papilla are seen two rings, the outer one darker and generally incomplete or absent, while the inner one is a faint white stripe, which becomes more marked as the patient grows older. The first is called the choroidal ring, and represents the edge of the choroidal coat of the eye where it is pierced by the nerve. The second is the scleral ring, which is the edge of the sclerotic coat. The centre of the optic papilla may be even with the surface or cupped, and may be stippled or dotted in appearance. The retinal arteries emerge from this central spot and the chief venous trunks empty into it. Generally one arterial and one venous stream pass up and a similar one downward, and both soon bifurcate, afterward still further dividing. The arteries are distinguished from the veins by their bright-red hue, while the veins are darker in color. The veins are about one-third larger than the arteries. A bright stripe due to an optical delusion seems to divide each vessel longitudinally into two parts. The arteries of the normal eye do not pulsate, but pulsation of the veins is quite common. It must be remembered that the appearance of the papilla and of the bloodvessels as they leave it varies very greatly within perfectly physiological limits. As already stated, the cupping of the papilla may be quite deep or quite shallow, and the bloodvessels may divide as already described, or divide in the papilla into four branches. The veins are usually more tortuous than the arteries. (Plate XXIII., Fig. 1.) The retina is practically transparent, so that the underlying choroid is seen. In persons with a dark skin the retina has a grayish hue in the neighborhood of the papilla, which is most marked on its nasal side and is slightly streaked.

To the outer side of the papilla, slightly below the horizontal meridian, is the macula lutea or yellow spot, which is about the size of the end of the optic nerve, but darker in color, somewhat granular, and devoid of any retinal vessel. It is the point of the eye-ground in which direct vision is best developed. In its centre is a bright spot, the fovea centralis. As a person grows older these clear distinctions vanish and the macula lutea is to be distinguished from the surrounding eye-ground only by its darker hue and the absence of vessels. The macula is difficult to see, because as the light falls on it the pupil at once contracts. If the eye is dilated by a mydriatic, however, and the patient looks directly into the ophthalmoscope, the macula is readily seen.

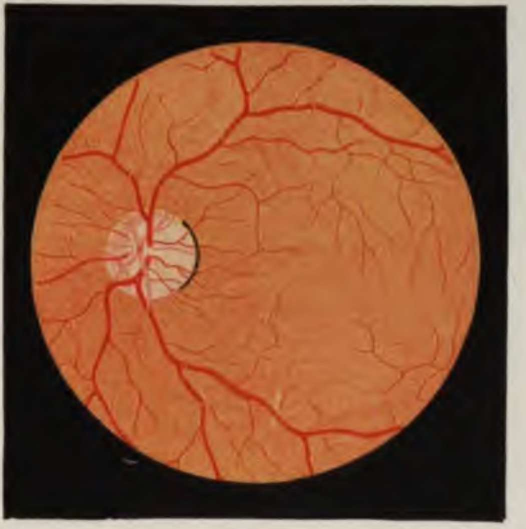
The red glare produced by throwing the light into the eye by the ophthalmoscope is due to reflection from the bloodvessels of the choroid coat.

The pathological significance of alterations in these normal appearances is very great. Let us suppose that on examining the eye-ground we find the end of the optic nerve red and its edges irregular and obscure, or, if the morbid condition is further advanced, that the nerve-head looks protruding or mound-like and the arteries going to it are smaller than normal and partly concealed, while the veins are enlarged and tortuous. Hemorrhages may be seen in the papillary region or near it, occurring in flame-like shapes. These are the signs of optic neuritis, and optic neuritis depends upon intra-orbital or intracranial disease, although, if the process is not marked, it may be due to hypermetropic astigmatism. Vision is often unaffected, but if the lesion be in the cerebellum sudden blindness may come on.

As some differences of opinion exist as to the various forms of neuritis of the optic nerve, the term papillitis is often used to signify all the forms of optic neuritis which we meet with, or in other cases is spoken of as choked disk. Papillitis is more commonly the result of brain-tumor than of any other intracranial lesion, and, again,

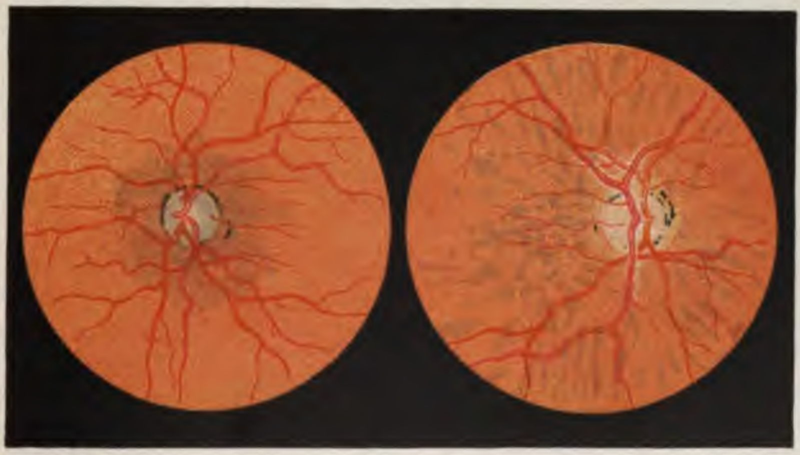
PLATE XXIII.

FIG. 1.



Normal Eye-Ground (average tint).
(Norris & Oliver.)

FIG. 2.

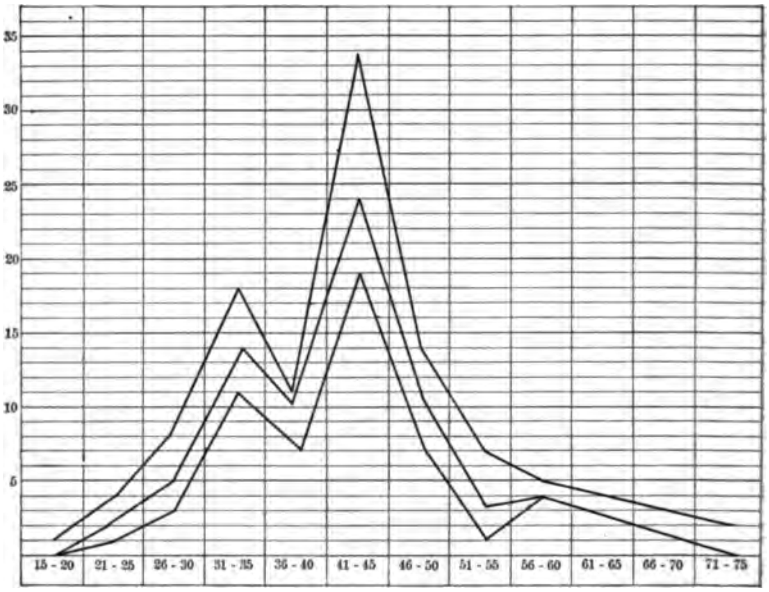


Primary Atrophy of Optic Nerve (Spinal Atrophy). Modified from Haab. (de Schweinitz.)

Post-Papillitic or Consecutive Atrophy of the Optic Nerve Modified from Juler. (de Schweinitz.)

the disk usually result from diseases in the optic centres or in the nerve itself. Thus there may be present a tumor pressing on the chiasm or optic tracts.

FIG. 217.



Upper curve, frequency of tabes. Middle curve, frequency of severe ocular symptoms. Lower curve, frequency of atrophy of the optic nerve. (BERGER.)

Again, if on the use of the ophthalmoscope we find that there is a faint haziness of the retina, that whitish streaks are seen in it which may be bluish-gray or yellowish in hue, that the bloodvessels are tortuous and minute vessels are easily seen because of their enlargement, that hemorrhagic exudations of a flame-like character are present and that dark pigmented spots show where previous hemorrhages have been, and, finally, that the head of the optic nerve is not clearly outlined, we have the picture of retinitis. Generally, in association with these signs, we find as subjective symptoms changes in the visual field, a distorted vision, so that straight lines appear bent inward or outward, and there are pain and fear of light. If in addition to these symptoms the vitreous humor is opaque, syphilis may be present, and the iris may give evidence of iritis. Where the hemorrhages are very manifest and profuse (hemorrhagic retinitis) the cause may be disease of the heart and bloodvessels.

By far the most important of these forms of retinitis from a diagnostic stand-point is what is known as albuminuric retinitis, or that due to Bright's disease. Here, in addition to the flame-like hemorrhagic areas, we find irregular spatterings of white which may be star-shaped. The importance of the discovery of such changes is that by it the first suspicion of renal trouble is aroused. This sign is of the greatest value in pregnancy. Retinitis also sometimes results from diabetes.

Hemorrhages into the retina without retinitis usually are the result of septicæmia, ulcerative endocarditis, hæmophilia, diabetes, gout, and malarial fever of a severe type. They are also seen in cases of great cardiac hypertrophy with stenosis, and after suffocation.

Ophthalmoscopic evidence of general arterial disease not infrequently manifests itself by œdema of the retina and retinal hemorrhages; but an early sign, which recently has been particularly pointed out by Mr. Marcus Gunn, is the influence of the arterial pressure on the venous blood-streams of the retina, where artery and vein cross one another. There may be simply inequality in the calibre of artery and vein, or the vein may be somewhat displaced, where it lies beneath the artery, in the direction of the arterial circulation and its flow obstructed. In advanced cases the vein is greatly narrowed where the artery crosses it and distended on its peripheral side. When these appearances are well studied they are exceedingly suggestive of early arterial changes. Changes of this character, as the author can testify from studies made by Dr. de Schweinitz, are of serious prognostic import and may be the forerunners of intracranial extravasations.

The iris indicates disease in other organs more rarely than the retina and optic nerve and the muscles, but an irregular pupil indicating an old iritis should raise a question as to a history of injury or rheumatism.

Finally, it should not be forgotten that cataract sometimes occurs as the result of diabetes mellitus and that corneal ulceration is often an evidence of scrofulous tendencies, while a distorted pupil due to an old iritis should raise a suspicion of syphilis.

It must not be forgotten that patients often have, in distinction from distorted images, visions or flames of light or bright sparks before the eyes, or in their place dark spots called muscæ volitantes. Often the visions are the prodromes of an attack of migraine or of an epileptic seizure. In the case of spots of light or stars we usually find them as a result of severe indigestion, and the dark spots may arise from the same causes. Muscæ volitantes may also be due to small particles of mucus floating over the cornea or to small floating bodies in the vitreous.

Partial or complete blindness is sometimes seen in cases which are under the influence of a drug, as, for example, quinine or other drugs; and sometimes partial or complete blindness results from uræmia (uræmic amaurosis). As a rule, it does not occur as a single symptom, but follows an attack of acute uræmic manifestations—that is, it is found after a convulsion or period of coma has passed by. As a rule, nothing abnormal is found in the eye to account for it, and the pupillary reflexes are intact. The effect of the poison in the blood is, therefore, exercised upon the optical centres, probably in the occipital lobe. Sight is usually regained in these cases in a few days.

PART II.

THE MANIFESTATION OF DISEASE BY SYMPTOMS.

CHAPTER I.

CHILLS, FEVER, AND SUBNORMAL TEMPERATURES.

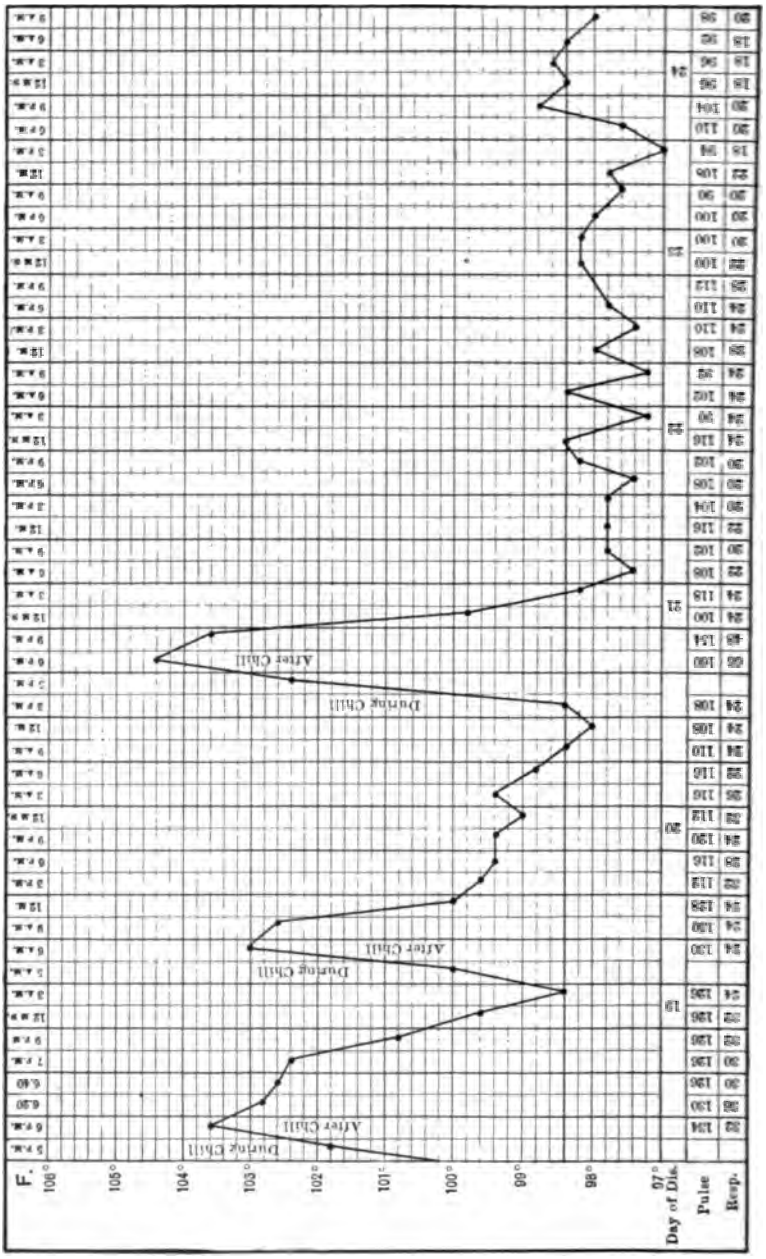
Chills—The methods of taking the temperature—The significance of fever—The febrile movements of various diseases.

A CHILL is of very considerable diagnostic importance when observed by the physician, or even when reported as having occurred in the immediate history of the patient. It may follow prolonged exposure to cold, without the subsequent development of disease, or be a precursor of some acute malady. Often it is an early symptom of the onset of one of the acute infectious diseases, such as croupous pneumonia, erysipelas, or scarlet fever. In other instances it is a symptom of the development of a purulent or pyæmic process. When chills recur repeatedly they may be due to malarial infection, in which case they may be controlled by using quinine; as a result of deep-seated abscesses and general pyæmia; and finally, they may indicate tuberculosis or ulcerative endocarditis. In some cases of typhoid fever a chill ushers in the attack, and chills repeatedly occur without apparent cause, so that the disease may be singularly like remittent malarial fever. (Figs. 217, 218.)

Fever is that state of the human body in which its temperature is raised above the normal limit, or 98.8° F., but variations from 97.8° to 99.5° may occur without indicating disease. From 99.5° to 100.4° the temperature is spoken of as subfebrile, from 100.4° to 101.3° as mildly febrile, while the term decidedly febrile is applied to temperatures varying from 103.1° to 105°. Hyperpyrexia is a term applied to a febrile movement in which the temperature rises as high as 106° F. Cases are on record of a temperature of 115° or even more.

The method of taking the temperature consists in placing a self-

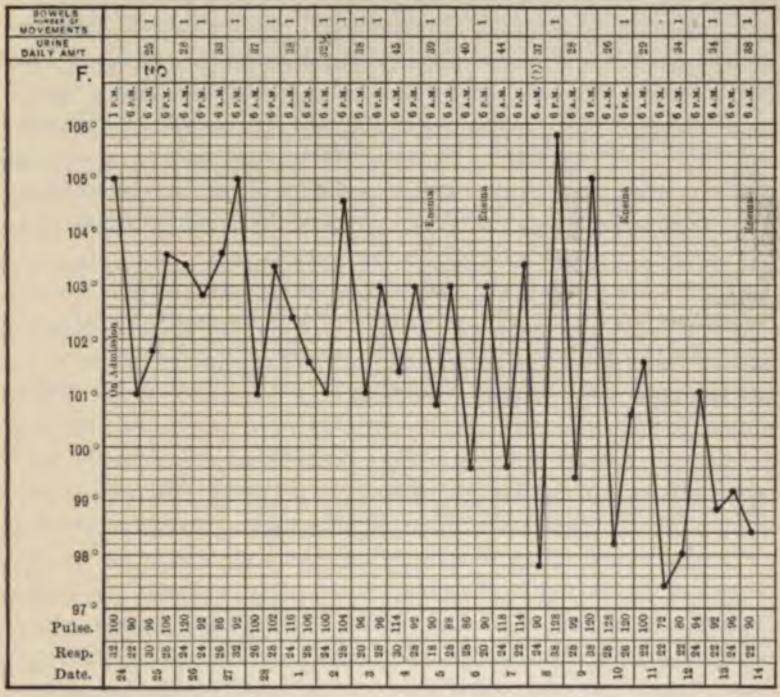
FIG. 217.



Toward the end of this case of typhoid fever severe chills developed on the 18th, 19th, and 20th days, and the fever ended by crisis.

registering clinical thermometer in the mouth under the edge of the tongue, the lips being then closed tightly about its stem; or of inserting it in the axilla, the hand and arm being then placed across the patient's chest, or epigastrium, so as to cause the axillary tissues to be in close contact with the bulb of the thermometer. Before the thermometer is placed in the axilla this space should be carefully wiped dry, since if perspiration is present its evaporation will so chill the thermometer that a false record will be made by the index.

FIG. 218.



"Chills and fever" in course of typhoid fever.

Sometimes the temperature of the patient is taken by inserting the thermometer into the rectum; and, if this is done, the bulb should be passed well inside the external sphincter. Rarely the temperature is taken in the vagina. In the rectum and vagina the normal temperature is about one to one-and-a-half degrees higher than in the axilla.

The precautions to be taken in all cases in which a thermometer is used, in addition to those named, is to have a thermometer which is accurate, and to be sure that there is no acute or chronic inflammatory process present which will produce local heat, and so give an erroneous impression as to the actual temperature of the entire body. This is particularly apt to be the case in diseases of the mouth in children: thus, stomatitis may raise the local temperature from one to two degrees. Hot liquids, if taken into the mouth just previous to or during the time at which the thermometer is inserted, will so raise the temperature of the local tissues as to make the thermometer register several degrees above normal, and a similar effect may be produced by cold liquids or ice held in the mouth. This subject has recently been studied by Lazarus-Barlow, who asserts that the effects of hot objects taken into the mouth last much longer than do those produced by cold, and that a mouth-temperature should never be taken within one hour of the time that any hot food is ingested. He even shows that holding the mouth open for some time renders a true estimate of the body-heat impossible, and advises that the temperature shall never be taken in the mouth if it is possible to take it elsewhere.

Febrile movements are generally associated with a dry, hot skin, but sometimes with a cold, wet skin. The latter condition is of evil significance, as a rule, and should be remedied if possible.

The significance of fever is great. It always shows the presence of an ailment sufficiently severe to make it wise for the physician to order the patient to bed till the fever abates or until he can surely determine its cause. The significance of a raised bodily temperature from a physiological point of view is that the nervous centres governing heat-production and heat-dissipation are disturbed by some substance circulating in the blood or by reflex irritation, or perhaps by both. The danger of high fever is that it may cause morbid changes in the protoplasm of the heart or in the vital centres at the base of the brain, but the danger of ordinary febrile temperatures has been greatly exaggerated. Indeed, in some cases moderate fever probably aids the body in combating or, rather, conquering the disease which has attacked it. This may occur in three ways, namely, by producing a temperature less favorable to the growth of certain disease-germs than is the bodily temperature in health; by increasing cellular activity it may increase phagocytosis and the development of antitoxic materials; and, finally, by virtue of the increased temperature, the effects of poisons may be rendered *nil*. This is the case, for example, in regard to the drug digitalis, which will rarely produce its ordinary effects on the heart when well-marked fever is present. Another point of importance in this connection is, that the duration of fever has more to do with its importance as a symptom than has its degree, for a temperature of 105° for a few hours may be borne with immunity, whereas one of 103° for many days cannot fail to produce evil effects.

Fever in children does not possess nearly as grave significance as it does in adults, for children often develop high temperatures from slight causes and have speedy recoveries. The balance of their heat-mechanism is easily upset. The older the patient the greater the significance of fever, and a rise of two or three degrees in a man of sixty years is more alarming than one of four or five degrees in a child of five or six years.

When fever is not due to a distinct pathological change in some part of the body, generally of an inflammatory kind, it may arise from so mild an irritation of a mucous membrane that a catarrhal condition is set up. Such fevers are seen in cases of mild gastro-intestinal catarrh in children after the ingestion of bad food or exposure to cold. Sometimes fever apparently arises as the result of the reflex irritation produced by difficult teething (see chapter on the Mouth and Tongue), although in many instances the fever of dentition depends upon a more or less closely related, but overlooked, gastric catarrh. Sometimes after a urethral sound or catheter has been passed into the urethra of a man, in the course of a few minutes or hours he develops a severe chill, followed by a fever which may be quite high, but does not last long.

Fever accompanied by apathy and listlessness is not of itself of grave omen, unless there be evidences of toxæmia producing interference with vital organs, but if such symptoms change into a state of active fever with delirium and jactitation the change is to be regarded as dangerous.

FEVER IN INFECTIOUS DISEASES.[1] Nearly all infectious diseases are ushered in by the development of fever of greater or less degree, and this is particularly true of the exanthemata. Inquiry should, therefore, be made by the physician as to the previous history of infectious disease. If one or more of the eruptive fevers have already been present, they can usually be excluded from the diagnosis of the illness at the time of the visit. If, on the other hand, there is a history of old pulmonary disease or acute articular rheumatism, this may indicate that another attack is coming on.

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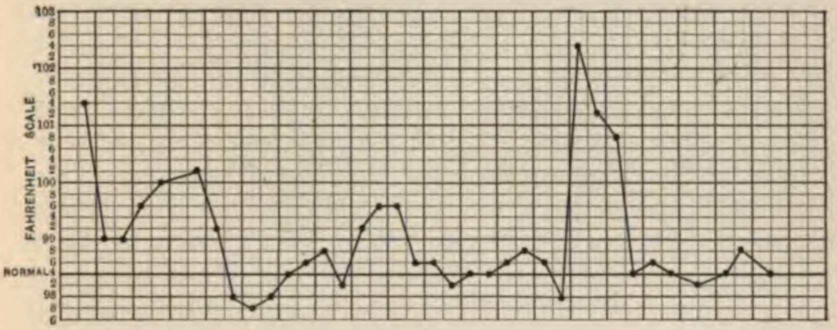
[1] In this connection the student should also read that part of the chapter on the skin devoted to the consideration of the eruptive infectious diseases.

In *typhoid fever* the febrile movement is very characteristic in some cases, although in many instances it does not follow the description laid down in text-books. After several days of general wretchedness the patient develops a slight fever of from 100° in the morning to 101° at night, and this temperature progressively rises so that the next morning it may be 101° and that night 102°, the next morning 102°, that night 103°, and so on until the morning temperature may be 103° and the evening temperature 104° or rarely 105°. The fever usually reaches its acme by the end of the first week or ten days, and then for another week remains practically unchanged, there being a morning fall and evening rise of an almost equal extent. Toward the end of the third week, or sometimes earlier or later, according to the severity of the attack, the morning remissions become more marked, and then the evening rises fail to reach their former height. Often these marked morning remissions are the first indication of the tendency to recovery. Very high evening temperatures are indicative of a severe attack, but are not so indicative of serious illness as are high temperatures in the morning. After the third week, in a moderately severe case, the temperature falls gradually till by the twenty-eighth day it usually reaches the normal. In very rare cases the temperature speedily reaches its acme at the very beginning of the disease, and then passes through the course already described. Such cases are generally prolonged, but may in some instances end by the fourteenth day. In other instances the development of high fever in the early stages of enteric fever, associated with severe general symptoms, is indicative of a short attack rather than a prolonged and severe one.

Sudden falls of temperature during the course of typhoid fever are nearly always of grave import. The most common cause of such a sudden fall is an intestinal hemorrhage, and the fall may occur sometimes before the blood appears in the stools. In other cases such a fall is an evidence of intestinal perforation. The other causes of a sudden fall are severe nose-bleed, or hemorrhage of any form; as, for example, that occurring in connection with abortion in a female patient. Sometimes, too, without any of these causes, the temperature falls very rapidly, and the patient goes into collapse. Such cases are very grave and the prognosis is unfavorable.

A recrudescence or return of the fever, in which it rises quite rapidly to a point as high or higher than at any time during the attack, occurs in some persons who, during the stage of convalescence from typhoid fever, take solid food too soon, or are excited by the visit of a friend. Such rises are but temporary. (Fig. 219.) More rarely, as a result of getting out of bed too soon, or bad feeding, or other cause, a true relapse takes place, and the disease runs a second course, which is usually, but not always, of a shorter and milder character than the first attack. (Fig. 220.) Sometimes a mild, irritative fever, perhaps due to anæmia, persists for some weeks, but the physician should not rest content with a belief that anæmia is the cause until he has excluded all possibility of there being pulmonary, pleural, acute renal, or bone disease, as these conditions very commonly ensue as sequels of typhoid fever. In other instances, after the morning temperature has reached normal, the evening temperature remains pyretic for a number of days, and this may persist for some time. In a number of instances I have known the use of strychnine in full doses, at this period of the disease, to produce ranges in temperature, which ceased as soon as the use of the drug was stopped.

FIG. 219.

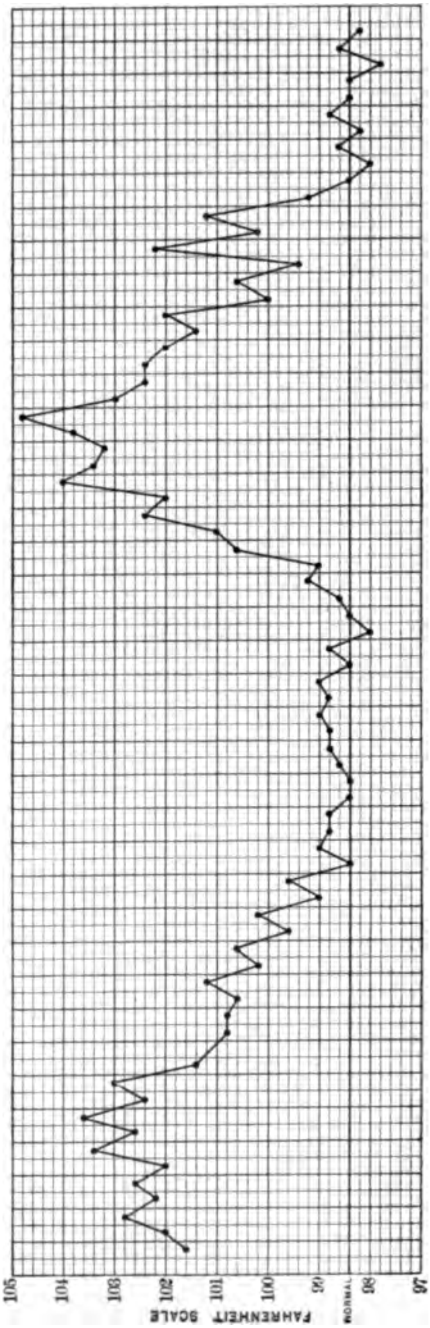


Showing recrudescence of fever in a case of typhoid fever.

If the temperature in the course of a case of typhoid fever rises as high as 107° or 108°, the prognosis at once becomes very grave.

Very rarely enteric infection, so called, runs its entire course without any fever. Fisk, of Denver, and others have seen such cases, and the author had five of them at St. Agnes's Hospital in one term of service.

Fig. 220.



Showing a relapse in typhoid fever.

Strümpell asserts that as a rare occurrence the fever in this disease may become intermittent, being normal in the morning and as high as 104° at night during almost the entire illness.

The association of such a temperature-curve as just described with the other characteristic signs of typhoid fever, as, for example, the development of the rose-rash on the chest and abdomen, on or about the seventh day (chapter on the Skin), the ochre-colored, loose stools, the peculiar stupid, drowsy appearance of the face, and in some cases the peculiar typhoid odor about the patient, all make the diagnosis certain.

The differential diagnosis of *acute tuberculosis* from typhoid fever may be quite difficult in certain cases. When the symptoms of the two conditions are compared this is not difficult to believe, for we often have in both diseases headache, epistaxis, a very similar temperature-chart, and a feeble pulse, while there may be in both conditions an eruption on the skin, which rather tends to confuse the physician than to aid him. Again, the delirium in each case is very similar, and the facial expression of the patient in both diseases is apathetic. Even the respiratory sounds in both diseases in their early stage may be apparently only those of a moderate bronchitis; and, finally, abdominal swelling, tympanites, and meteorism may occur in both maladies. Under these circumstances the hereditary and recent history of the patient may be of much value, as showing a tendency to tuberculosis on the one hand, or exposure to typhoid infection on the other. Again, if it be typhoid fever, the spleen is nearly always found on percussion to be enlarged. Then, too, the lesions in the lungs of a typhoid-fever patient are generally at the bases, while in tuberculosis they are oftener at the apices. The stools may be loose in both diseases, but in typhoid fever they are apt to be ochre-colored; and, again, in tuberculosis the loss of flesh is often exceedingly rapid, and profuse sweats and high fever are frequently seen. The mental apathy in typhoid fever is more marked, as a rule, than it is in tuberculosis. The finding of Widal's reaction in the blood, or the discovery of the bacillus of Eberth in the feces and the presence of the diazo-reaction in the urine, would, of course, indicate typhoid fever. (See chapter on Urine.) An absence of leucocytosis would also indicate typhoid fever, for this condition is usually present in acute tuberculosis. Finally, careful and repeated examinations of the chest will usually, in the course of the disease, demonstrate the presence of tuberculosis of the lungs or bowels, if this be the cause of the illness. It seems hardly necessary to state that if any expectoration exists the sputum is to be carefully examined for tubercle bacilli in all doubtful cases, but while their presence proves tubercular infection to be present, their absence does not prove the absence of this infection.

Irregular forms of malarial fever, particularly those forms due to infection by the æstivo-autumnal parasite (see chapter on the Blood), may closely resemble typhoid fever. In many instances such cases are diagnosed as typhoid fever, and probably some cases of true typhoid fever are thought to be malarial fever. The following differential table, drawn up by Thayer, is of interest in this connection. There is no such disease as typho-malarial fever, although there is no doubt, whatever, that pure typhoid infection may result in the production of a fever which closely follows the remittent and intermittent malarial types, and which is often associated with so much gastric disturbance and vomiting, and so lacking in the more prominent typhoid symptoms usually seen, that the picture of remittent malarial fever is clear, while the true picture of typhoid fever is clouded. Again, there can be no doubt that cases of true malarial infection occur in which the symptoms so closely resemble those of typhoid fever that a purely clinical diagnosis is almost impossible, particularly if an epidemic of typhoid fever is in full swing at the time. Finally, there can also be no doubt that it is possible for the patient to have a double infection with the bacillus of Eberth and the plasmodium of Laveran, in which case, however, the malarial manifestations are usually dwarfed by the typhoid poison, and only are marked at the onset of the enteric fever and its termination. To this mixed infection the term typho-malarial fever may be correctly applied to indicate not a separate disease, but a double infection. Etymologically, this term might also be used to define a condition of malarial fever in which, because of profound debility, the patient was in a typhoid state—that is, in a condition of which typhoid fever is a type. Practically, however, it should be discarded or limited in its use to the double infection just described.

Remittent Fever.	*Typhoid Fever.*
Onset generally intermittent.	Onset gradual and progressive.
Irregular remissions.	Regular, though very slight morning remissions with evening exacerbations of temperature.
The temperature may arrive at 40° C. (104° F.) within twenty-four hours.	The temperature does not reach 40° C. (104° F.) before the third or fourth day.

Remittent Fever.	*Typhoid Fever.*
Headache rare in the beginning; of a neuralgic character, pulsating, variable in its position and intensity. Sclera subicteric from the onset.	Headache from the beginning, permanent, severe, frontal. Sclera white.
The apathetic expression of the face, the dryness of the tongue, and sordes upon the teeth are not very marked.	These symptoms are well marked and progressive.
Breath foul.	Breath has a peculiar mouse-like odor.
The delirium may come on in the early days; it is recurrent, but changes with the exacerbations of temperature and other symptoms, and may give way to grave symptoms related to other organs.	Delirium appears only when the disease is well pronounced; it is often persistent, and variable only in degree.
If there be pulmonary congestion, the cough and other symptoms come on suddenly; the areas affected change from one to the other lobe or lung, and may disappear and reappear again with varying intensity; dyspnœa is very pronounced; circulatory disturbances are marked, even syncope.	Pulmonary congestion is gradual and persistent; always hypostatic (the bases and dorsal surfaces of the lungs); the dyspnœa is less pronounced and later in appearing, depending more upon the abdominal conditions (tympanites, etc.).
There are usually restlessness and anxiety (jactitatio corporis).	There are usually relaxation, prostration, and stupor.
Peculiar grayish color of skin; sometimes a slight jaundice.	No jaundice.
Herpes common.	Herpes rare.
Anæmia more or less marked early in the course.	Anæmia absent, excepting in later stages.
No characteristic exanthem; urticaria not uncommon.	Characteristic roseola.
At times there may be transient tympanites or ileo-cæcal gurgling; they are but slightly pronounced and paroxysmal; diarrhœa is slight or absent, and has not the characters of that in typhoid fever.	Tympanites, gurgling, and diarrhœa appear slowly and may become well marked.
No distinct course.	Has a fairly characteristic course.
Urine high-colored; may show a trace of bile; Ehrlich's diazo-reaction rarely present.	Urine high-colored; bile absent; diazo-reaction present during the height of the process.
Blood shows no leucocytosis; eosinophiles not notably diminished; serum does not cause agglomeration of typhoid bacilli (Pfeiffer, Durham, and Widal); malarial parasites and pigmented leucocytes present.	Blood shows no leucocytes; eosinophiles diminished or absent; serum causes agglomeration of typhoid bacilli; malarial parasites and pigment absent.
Fever disappears under quinine.	Fever uninfluenced by quinine.
Is an endemic disease occurring particularly in rural districts; rarely epidemic.	Usually epidemic; prevailing commonly in cities.

The febrile movement and other symptoms of enteric fever are often imitated very closely by those of *ulcerative endocarditis.* In addition to an irregular fever, there may be diarrhœa, parotitis, stupor, and progressive feebleness in both diseases. An examination of the heart may reveal the presence of endocardial murmurs, which in association with signs of sepsis, or the existence of some focus of infection, such as a wound, a septic process, or the fact that the patient is in the puerperium, will render a diagnosis possible. (See also page 517.)

An irregular fever with muscular pains and a great deal of discomfort in the belly, the case simulating typhoid fever, may occur in cases of *trichinosis*.

A febrile movement closely resembling that of typhoid fever, a resemblance which is increased by the association with it of headache, insomnia, and anorexia, may be *Malta fever*, a disease which can be excluded in the vast majority of cases if there is no history of exposure to the exciting cause in the island of Malta, although it is not to be forgotten, that Malta fever has been met with in persons returning from Porto Rico and elsewhere during the last few years. Sometimes it may be confused with relapsing fever, except for the longer febrile movement. Thus, after three or four weeks of illness convalescence seems to be established, and the temperature falls, but in a few days all the symptoms return with even greater vehemence than before. Such relapses may occur again and again. Violent pain in the joints on moving the body is often present. In doubtful cases the agglutination test with the blood may be performed, if it is possible to obtain cultures of the *Bacillus melitensis*.

The temperature-chart of *typhus fever* is so different from that of typhoid fever that it gives us a valuable differential point at the very beginning of the disease, for, after several days of languor, headache, and pain in the limbs, the fever suddenly springs on the patient, so that on the first night it may reach 105° F. Often it reaches 106° in a day or two, and while present is constant, the morning fall being very slight indeed. The development of the spots in a copious eruption on the third to the seventh day, which spots may develop into petechiæ before fading, or remain unchanged in appearance, the great exhaustion, the severity of the illness, and the sudden rise of temperature, followed by a constant fever, point to typhus fever. Finally, the conclusion of the febrile movement in favorable cases by the end of the second week, by crisis or by a more rapid fall of temperature than we are accustomed to see in typhoid, all help to make the differential diagnosis, which is, however, in many cases very difficult or impossible in the early stages.

The temperature of *relapsing fever* nearly always rises suddenly at the beginning of the attack to from 103° to 105°, and remains high with slight morning remissions from three to seven days, when it suddenly falls as by crisis to the normal or below it, after being on the preceding afternoon or evening unusually high. Sometimes it falls as low as 92° or 93° F. The patient now remains free from fever for from several days to two weeks, when with a sudden leap the fever and other symptoms of the first attack recur. A temperature of 105° to 106° in relapsing fever rarely indicates a grave outlook, as it does in typhoid. The only condition which resembles this temperature range of relapsing fever is intermittent malarial fever; but the rarity of relapsing fever in America, the frequency of malarial fever in certain parts, the presence of the spirillum of Obermeier in the blood in relapsing fever, and the malarial germ in the blood of intermittent fever, all make the diagnosis possible.

In *scarlet fever* the temperature suddenly rises on the first day to 104° to 105° F., and still higher on the next day, and then remains constant as long as the eruption is on the skin in full development. Just so soon as the eruption begins to fade the temperature also falls, not by crisis, but by a lysis; not so slowly as in typhoid fever, but far more slowly than in pneumonia. This arrest of the fever usually takes place in simple cases by the end of seven days; and if it persists longer, is probably due to some complications, such as otitis, or the "collar of brawn," due to enlarged cervical glands. (Fig. 221.) The characteristic strawberry punctated rash and scarlet hue appearing on the first or second day, the ultimate dermal desquamation, the violence of the onset of the symptoms, the sore throat, and the peculiar appearance of the skin, all complete the clinical picture, particularly if the symptoms be in a child. (See chapter on Skin.)

FIG. 221.

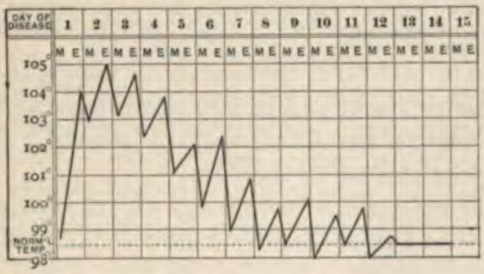
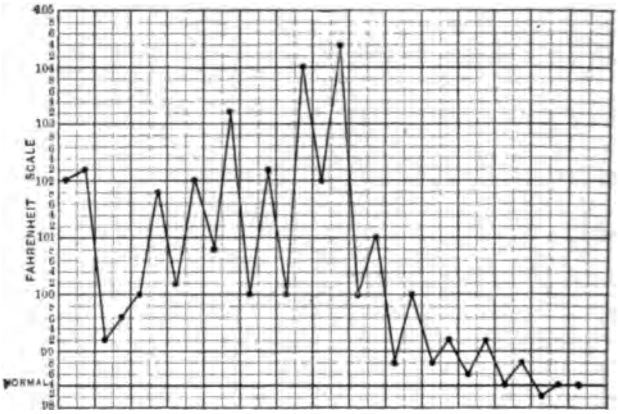


Chart of scarlet fever.

In rare cases the fever in scarlatina is remarkably mild or almost absent, and these cases, as a rule, have a favorable prognosis. If the temperature be very high and persistent, on the other hand, the case is usually to be regarded as most grave.

In *measles* the fever at first rises sharply to 103° or thereabout, then falls to a little above normal, is slight for several days, and then markedly increases with the development of the eruption on the fourth day, often ranging as high as 104° or 105°, at which point, with little variation, it remains for the two days during which the rash is well developed. (Fig. 222.) With the fading of the rash the temperature also falls by crisis. If fever persists to any extent, it is always due to some complicating cause other than the original disease; such a complication, for example, as a bronchial or gastro-duodenal catarrh.

FIG. 222.



Showing initial fever with the subsequent fall and then a rise when the rash is well developed in a case of measles. Also shows an ending of the fever by crisis.

The fever of *rötheln*, if any occurs, is very seldom more than 102°, and has no typical preliminary rise as has measles, so that the temperature-chart of the disease may aid materially in a differential diagnosis. (See chapter on the Skin.)

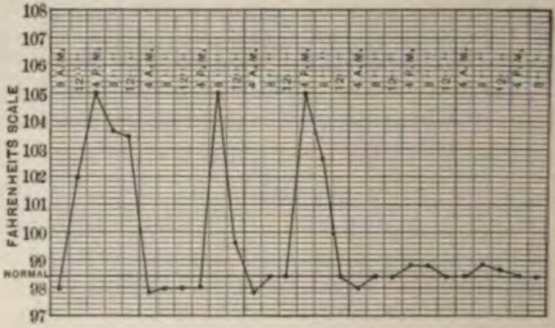
The febrile movement of *smallpox* is with the exception of that of typhoid the most characteristic of all the eruptive diseases. With a sudden onset of fever, pain in the back, severe headache, and malaise, the patient takes to his bed if possible, and his temperature if taken will be found speedily to rise even to 105° or more in some cases, and then falls back to almost normal for two or three days, during which time the eruption appears. In this way, therefore, the temperature-chart of variola differs diametrically from that of the eruptive fevers so far discussed, for in these cases the fever rises with the appearance of the eruption, whereas in this instance the temperature falls with the appearance of the eruption. This lower temperature persists for several days, from half to one degree above normal, till the ninth day of the disease or the sixth of the eruption, when with the change of the pocks from vesicles to pustules the temperature rises again in what is called the fever of suppuration, which lasts with greater or less persistence for at least a week, when it ends by lysis or a gradual fall. Excessively high fever of 108° is a sign of approaching death or at least of very grave import.

The febrile movement of *varicella*, or chickenpox, is usually of very short duration and of little severity; but it may reach proportions entirely out of consonance with the general systemic disturbance, which is usually very slight in previously healthy children. Thus it may rise in children who are prone to active febrile movements to as high a point as 105° for a very brief period, and yet may not seem to render the child ill.

The temperature-range seen in cases of *erysipelas* is quite typical. At the beginning of the attack the rise is quite prompt and sharp to 105° or 106° or even above this, and, instead of remaining constantly high through the course of the inflammatory process in the skin, goes through marked intermissions or remissions, which frequently occur and are followed by rises in temperature as high as that which occurred with the first onset. The fever ends in some cases by crisis, and in others by lysis, the latter mode of ending usually taking place in those cases which have had a very severe attack prolonged in character, or which have been in an asthenic state prior to the disease. The diagnosis of erysipelas is easily made by the brawny, swollen, and red skin, with the peculiar line of demarcation at the edge of the swelling. (See chapter on Skin.)

A fever which rises sharply from normal to 103° or 104°, being preceded by a chill and followed in a very few hours by a sweat, the whole term of acute illness, if we exclude general physical discomfort, lasting but eight to twelve hours, is in the majority of cases that of *intermittent malarial fever*. The peculiarities of intermittent malarial fever, aside from those just named, are that the febrile movement begins to decline before the stage of sweating begins, and in some cases it begins to rise before the sensation of chilliness of the first stage leaves the patient. (Fig. 223.)

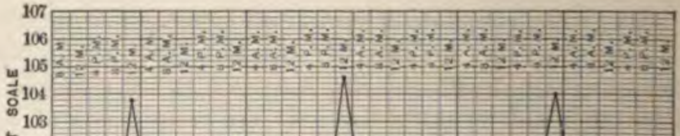
FIG. 223.



Showing daily paroxysm due to double tertian infection. One set of parasites segmented at 8 P.M. and the second set at 4 P.M. Paroxysm stopped by quinine on fourth day.

The fall of temperature is usually less abrupt than the rise, and is sometimes delayed by slight temporary rises or arrests in its downward course. The febrile movement is repeated at intervals, ranging from one to seven days or even at longer intervals than this. If the attacks occur daily, they are called quotidian, and this is due

FIG. 224.



Showing paroxysms of tertian fever, the segmentation of the organism occurring at about twelve o'clock every other day.

to infection by two sets of tertian parasites which undergo segmentation on alternate days, or it may be due to infection with three sets of quartan parasites. If the attacks occur every other day, they are called tertian (Fig. 224); if on the third day, quartan; if on the fourth, quintan. If two attacks come on the same day, it is called double quotidian.

Another point of importance in connection with malarial attacks is that they often occur earlier each day by an hour or more. Rarely, they are delayed.

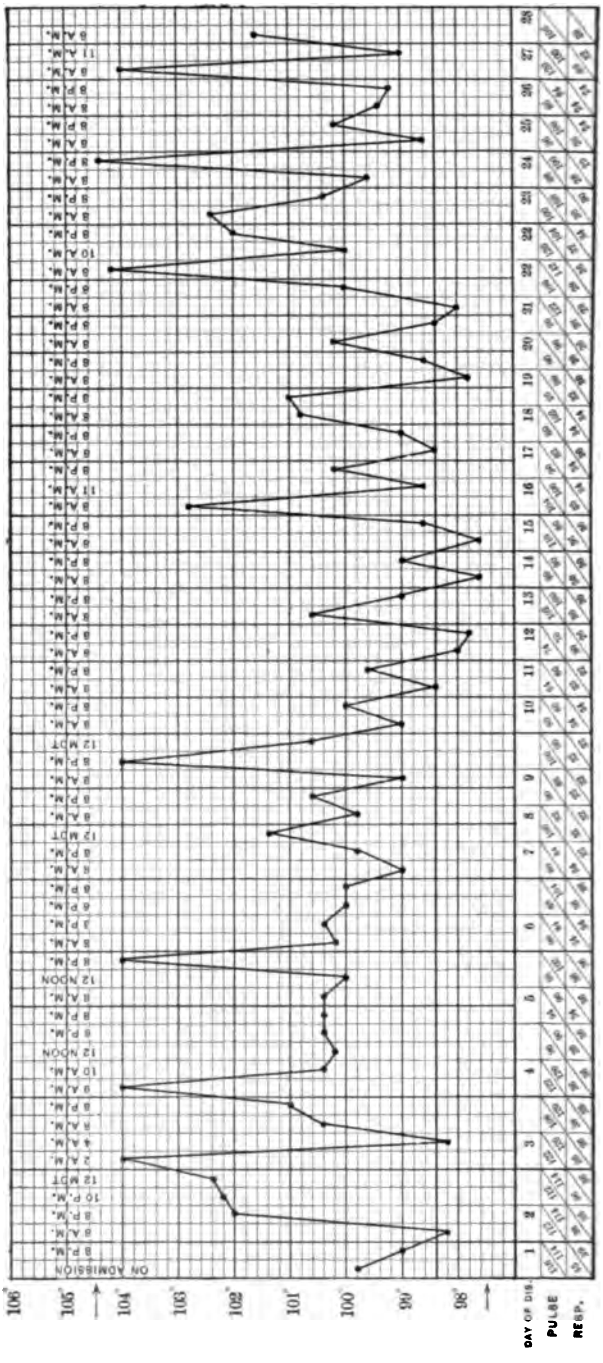
Intermittent malarial fever is to be separated from other intermitting fevers by a number of facts. First, the presence of the malarial organism in the blood at the time of the attack, or evidences of its presence at other times. (See chapter on Blood.) Second, by the history of exposure to malarial influences. Third, by the marked effect for good on malarial fever produced by the administration of quinine.

As stated in the chapter on the Blood, an examination of this fluid will reveal in practically every form of infection, except malaria and typhoid fever, an increased leucocytosis; but in malarial infection the leucocytes are not increased in number.

Care must always be taken that the intermitting fever of the various forms of sepsis is not diagnosed as malarial intermittent fever. The most common error of this character is the making of a diagnosis of irregular malarial intermittent, because chills, fever, and sweat appear every evening, when, in reality, the real cause is an undiscovered pulmonary or abdominal tuberculsois. Again, acute, ulcerative endocarditis and purulent phlebitis may cause similar symptoms, as may also hepatic abscess, impaction of gallstones, with suppurative cholangitis, causing the so-called Charcot's fever (see below). The absence of a history of malarial exposure, the possible presence of a cough, and the discovery of a tubercular lesion in the chest or abdomen by careful physical examination will aid in deciding that the fever is tubercular and not malarial in origin. (See chapters on Thorax and on Abdomen.)

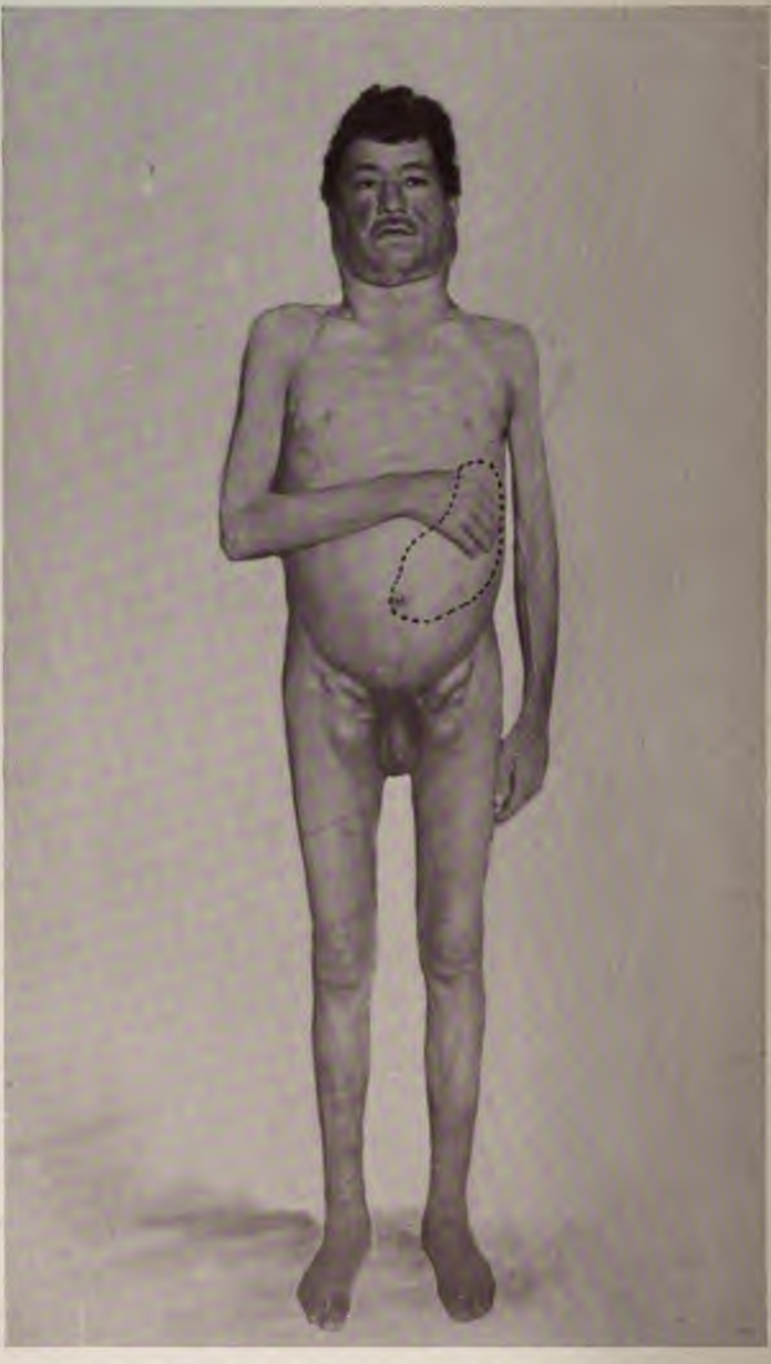
In *ulcerative endocarditis* the temperature-curve may exactly resemble intermittent malarial fever; but in many instances the presence of an external wound, acute sepsis in some part of the body, or the presence of the puerperium will reveal the source of an infection. (Fig. 225.) In the typhoid type of ulcerative endocarditis the profound asthenia and general prostration will separate the diseases even if the temperature-chart be useless. In this form the febrile movement is rarely typically intermittent. The crucial test of the differential diagnosis lies in an examination of the heart, in which a murmur may be heard in some but not in all cases, unless there has already been some grave valvular mischief. The cardiac feebleness and asthenia, on the one hand, and the result

FIG. 225.



Showing temperature-curves in a case of ulcerative endocarditis. (From a case in author's wards.)

PLATE XXV.



Hodgkin's Disease. (De Forest's case.)

of the blood-examination, on the other, aid the diagnosis. The duration of the case is not of much value in making a diagnosis, for cases of ulcerative endocarditis have lasted from two days to more than a year. Rarely it lasts more than six weeks. Death usually occurs in ulcerative endocarditis, unless there has been previously present chronic endocarditis, in which case recovery may rarely occur.

The discovery of some spot showing a *phlebitis* may point to this cause for intermittent fever.

The fever of *catarrhal* or *suppurative cholangitis* often closely resembles intermittent fever, but the presence of hepatic symptoms, of marked jaundice, of a history of gallstone colic, and of exceedingly severe rigors, enables us to separate them. In obscure cases the malarial organism should be searched for, and if the condition be one of cholangitis an examination of the blood will probably show leucocytosis. (See chapter on Pain, Gallstone Colic.)

When fever of an intermittent type has been observed, and intermittent malarial fever, tuberculosis, and cholangitis cannot be discovered as a cause, search should be made for tenderness and swelling of the liver due to *hepatic abscess.* Profuse sweats also may be found in such cases, as in most instances of septic fever. The diagnosis of hepatic abscess will be strengthened if there is a history of the patient having suffered from dysentery, as hepatic abscess is sometimes caused by amœbic dysentery.

The presence of fever preceded by chills, the temperature rising to 104° or even 105°, followed by excessive sweats, in a person who is profoundly cachectic, may be due to *pernicious anæmia* or to *septic poisoning,* as already pointed out; and it should be recollected that such a temperature-chart is sometimes seen in cases of gastric cancer. Similar symptoms as to fever in association with enlargement of the lymphatic glands, particularly those of the neck, indicate Hodgkin's disease (see chapter on Blood), or even more commonly tuberculous adenitis, which, however, is usually met with in the young and involves the glands near the jaw, while in Hodgkin's disease the glands near the clavicle are affected. Further, in Hodgkin's disease the swelling is usually bilateral, and to be found elsewhere than in the neck. (Plate XXV.) Again, in tubercular disease these glands often suppurate. The presence of the tubercle bacillus in an excised piece of the swelling will decide the diagnosis. In the opinion of some clinicians the two states are identical in cause.

An intermittent fever may also be seen in *suppurative pyelitis*, in association with pyuria. This pyelitis may or may not be tubercular.

Remittent fever rising and falling every few days for two or three weeks, rarely rising above 103° to 104°, and even falling to the normal line, associated with enlargement of the spleen and liver, yellowing of the skin, or jaundice, bilious vomiting, and a history of exposure to malarial poisoning, is characteristic of *remittent malarial fever*, a form more chronic and very much more grave than the intermittent form just described, because it responds less readily to treatment; and, secondly, because it is accompanied by more marked changes in the viscera. It depends upon infection with the æstivo-autumnal form of the malarial parasite. The conditions produced by this parasite are collectively grouped under the names remittent, continued, bilious remittent, and typho-malarial fever, or malarial fever of a typhoid type. In some cases the temperature and other symptoms will so closely resemble those of typhoid fever that nothing short of an examination of the blood can decide the diagnosis. (See chapter on the Blood.)

Care should be taken to recollect the fact that when typhoid fever develops in a young child the temperature may be so markedly remittent that an erroneous diagnosis of malarial infection may be made. In other words, "infantile remittent fever" is really typhoid fever in many instances.

A febrile process somewhat closely resembling remittent malarial fever, yet so rare, comparatively, as never to be confused with it, is *Weil's disease*. In this condition the fever runs a remitting course, is associated with jaundice and swelling of the liver and spleen, and the stools may be clay-colored. There is one important point of difference between malarial remittent fever and Weil's disease, namely, that in the latter gastro-intestinal symptoms are nearly always wanting or are mild, whereas in the former they are apt to be marked. Usually the fever of Weil's disease ceases by the end of two weeks or earlier. It is probably an infectious jaundice.

In *dengue*, a disease seen most commonly in epidemics in certain parts of the southern United States, the patient, after suffering from violent aching pains in the body and limbs, swelling of the joints, and the development of a variable rash on the chest, develops an active fever, which lasts with the pain till the fifth day, when both the pain and fever decrease or cease, and then often return with equal force. These facts, combined with the fact that it is an epidemic disease, separate it from malarial fever. Dengue and influenza, of an epidemic type, closely resemble one another, but in dengue there is rarely marked involvement of the respiratory tract as there is in influenza; there is an eruption which is not seen in influenza, and it is not followed or accentuated by such grave complications as we see in the more severe cases of influenza. Dengue is a disease of the South and influenza one of the North.

The fever of *yellow fever* is rarely over 103° or 104°, and is one of the milder symptoms of the disease; but it possesses this peculiarity, namely, that after the lapse of from twelve hours to several days there is a marked remission of the fever and all the other symptoms, and from this time on the patient may get well, or after a few hours this calm stage is followed by the true violent symptoms of the disease, such as black vomit, tarry stools, jaundice, and hemorrhages from the mucous membranes. Generally the full course of the disease to convalescence or death is run in about one week.

There are only two other diseases which can be readily confused with yellow fever, namely, dengue and bilious remittent fever. Dengue has been confused with yellow fever many times, and even the most experienced physicians have had great difficulty in separating them when the yellow fever has been mild. The most important points in their differentiation are the facts that in dengue there is usually a second onset of fever several days after the first onset, whereas this does not occur in yellow fever. Again, the eruption on the skin is not seen in yellow fever, and a rapid pulse is present in dengue, whereas in yellow fever the pulse is usually not very rapid. On the other hand, in yellow fever we usually meet with jaundice, albuminuria or suppressed urine, and a hemorrhagic tendency of a marked degree, all of which are absent in dengue. Further, death from dengue is very rare.

A case of bilious remittent fever occurring during an epidemic of yellow fever is almost certain to be incorrectly diagnosed. In the absence of an epidemic, however, the probabilities of the case being bilious remittent fever are very great, and the presence of bilious vomiting rather than that of blood, the characteristic temperature-chart, and, above all, the presence of a history of malarial exposure and of the signs of malarial infection in the blood, with the partial control of the symptoms by quinine in certain stages of remittent fever, point to the diagnosis of malarial disease rather than to yellow fever.

Stubbert gives the following differential tables of these fevers:

Yellow Fever.	*Pernicious Malaria.*
Headache bilateral-frontal, and post-orbital.	Headache generally unilateral-frontal, and temporal.
Temperature and pulse divergent; temperature rarely higher than 104° F.	Temperature and pulse correlative; temperature generally 105° to 107° F.
Albumin present in large quantities early in the disease.	Albumin rarely present.
Quinine has no effect on the progress of the disease.	Quinine has a specific effect if given hypodermically and early.
Stage of remission on third or fourth day.	Remission not present.
Attacks new arrivals.	Generally history of chronic malarial infection.
Always history of exposure to infection.	No history of exposure to infection.
Black vomit appears on third or fourth day.	Black vomit appears within thirty-six hours.
Hematuria very rarely present.	Hematuria a marked symptom.
Liver unchanged.	Liver enlarged and tender.

Yellow Fever.	*Dengue Fever.*
Cephalalgia and nephralgia are characteristic and constant.	Pain most severe in joints and muscles, and is *paroxysmal.*
Pulse and temperature divergent.	Pulse and temperature correlative.
The slowing of the pulse begins early in the disease.	The slowing of the pulse occurs late in the disease.
Congestion of face early in the disease. No edema.	Rash on face, followed quickly by edema.
Albuminuria.	Albuminuria absent.
Icterus.	Icterus absent.
Black vomit.	Black vomit absent.
No eruption.	Polymorphous eruption, followed by desquamation.

Just as in yellow fever, so in spotted fever or *cerebro-spinal meningitis* of an epidemic form, the fever itself is one of the least important symptoms, for, aside from the fact that it is apt to be irregular and intermitting, it is rarely very high, as compared with the violent cerebro-spinal symptoms, the rigidity of the back of the neck, the headache, convulsions, and vomiting. The presence of these symptoms in an epidemic does more to confirm a diagnosis than the febrile movement. In some cases of spotted fever, however, of a very grave type, the fever becomes a hyperpyrexia, but in cases tending toward recovery the temperature usually begins to fall by lysis before any moderation in the other symptoms is manifested.

When the physician feels it necessary to determine the true character of the affection without delay, he may resort to lumbar puncture. An ordinary aspirating needle is introduced between the second and third, or third and fourth lumbar vertebræ (Fig. 54), and by this means some of the cerebro-spinal fluid is obtained, which should be received in a sterile test-tube, and examined microscopically for the characteristic diplococci of spotted fever, and for the other micro-organisms. The needle should be inserted 4 centimetres (1½ inches) in children, and 6 to 8 centimetres (2 to 4 inches) in adults, to reach the fluid around the cord. It is not a dangerous operation. Pfaundler, on the other hand, recommends that the puncture be made in the lumbo-sacral space, and that it should be performed while the patient is in the sitting position. (For area for puncture see figures on the spinal column in chapter on Feet and Legs.)

He believes that very high pressure of the fluid in the spinal membranes is present in tuberculous meningitis, and that this high pressure is so rare in other conditions that its presence is of diagnostic value. It is found, however, when the patient is suffering from purulent meningitis, from spinal tumor, and in some cases of functional neurosis. On the other hand, normal pressure excludes the presence of meningeal or cerebral affections. If the fluid which is obtained contains disintegrated blood, the patient is probably suffering from pachymeningitis or injury. If, on the other hand, the blood is fresh in its appearance, its presence is probably due to the puncture.

A very important point in this connection, insisted upon by Pfaundler, is that if the fluid is clear, every inflammatory affection of the meninges, except tuberculosis, may be excluded from the diagnosis. In tuberculosis, it is often cloudy, and in the later stages of the disease nearly always so. If the fluid is purulent, it indicates the presence of purulent or epidemic meningitis. A clear fluid is found in health, in serous meningitis, hydrocephalus, and functional neuroses, and it is generally clear in cases of tumor, uræmia, and sepsis. The albumin present varies from 0.02 to 0.04 per cent., and if it is in excess of 0.05, it is probable that the disease is an inflammation or tumor; if it is present in still larger amounts, it is probably tuberculous meningitis. If, again, on analysis of the fluid the normal sugar which it contains should disappear, there is probably an inflammatory process present, and therefore if sugar is present an inflammation can be to a certain extent excluded.

Even in the presence of an epidemic of spotted fever it should never be forgotten that middle-ear disease often causes marked meningeal symptoms, and that croupous pneumonia often produces a similar train of manifestations, probably by infection with its particular micro-organism. The possibility of tubercular infection producing such symptoms should cause the physician to examine the patient carefully for signs of tubercular disease in other parts of the body from which infection might arise, as, for example, the lungs.

The fever due to *septicæmia* may produce a temperature-chart which closely resembles that of enteric fever, but septic fever generally possesses one characteristic which, in the face of other symptoms suggesting sepsis, is of great importance, namely, the extraordinary rises from normal to 105° or 106°, and from that point even to a subnormal degree within a very few hours, so that the lines on the chart pass up and down in long sweeps. These sweeps are even more sharp and sudden than in an intermittent malarial fever, and their cause is determined by the discovery of some septic process in some part of the body. The presence of such a chart, in association with dull or violent headache, delirium, vertigo, and vomiting independent of taking food, would point to cerebral abscess, particularly if a history of injury could be obtained.

A somewhat similar chart to this may occur in connection with cases of *active pulmonary tuberculosis*, when the lesions are well developed and septic absorption is active; but usually in the hectic fever of phthisis we have an approximately normal morning temperature, with a rise from two to three degrees, or even more, toward night. (Fig. 226.)

This symptom of fever in any form occurring in a person with suspiciously "weak lungs" should cause the physician to be confident that he has overlooked some focus which another careful examination may discover, and it possesses another important diagnostic

FIG. 226.

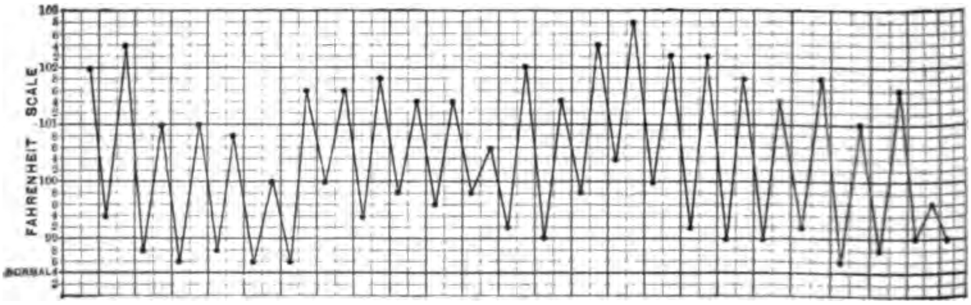


Chart of a case of pulmonary tuberculosis, showing rising and falling of the temperature morning and night.

significance, namely, that the more active the febrile movement in phthisis pulmonalis the more active the disease-process, and the less active the fever the less active the process. Fever may, however, be almost entirely absent in some tubercular cases with extensive disease of the lungs.

The febrile movement of *acute miliary tuberculosis* has nothing characteristic about it, except that in some cases it may closely resemble that of typhoid fever, and if the physician does not carefully examine the case an erroneous diagnosis may be reached. If, however, the disease involves the meninges of the brain, a hyperpyrexia may be developed, and death speedily occurs in such cases, either in the fever or in a sudden collapse. The peculiar dyspnœa, the cyanosis, the profuse sweats, and the diffuse pulmonary signs render a diagnosis of acute miliary tuberculosis possible in some cases.

When fever is associated with marked catarrhal symptoms, chiefly of the bronchial tubes and upper respiratory tract, with sneezing, lassitude, pains in the back and limbs, and excessive cough, the fever rising as high as 104° or 105° in severe cases, and then falling almost to normal, we may have before us *influenza* or catarrhal fever, either of the sporadic or epidemic form. In this condition there may be in severe cases great prostration and cardiac failure or vomiting and diarrhœa. The febrile movement is of the most irregular type, even when some grave complication, such as severe bronchitis or pneumonia, comes on, although croupous pneumonia rarely occurs as a complication of "la grippe."

The respiratory symptoms just described are also seen frequently in association with moderate fever, in "*hay fever*" that condition seen in susceptible persons during the haying-season or late in the summer.

The fever of *croupous pneumonia* of the croupous type runs a very typical course in uncomplicated cases. Following a more or less severe chill, the fever quickly mounts to the high point of 103° or 104°, or even more than this. (Fig. 227.) For the next few days, if not modified by the antipyretics and the use of cold, the fever remains high; but there may be temporary remissions which look as if crisis was about to be established, when in reality they are followed at once by a return of the fever

FIG. 227.

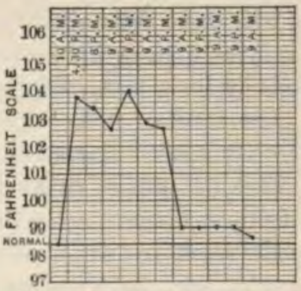


Chart of a case of croupous pneumonia, showing primary rise of temperature to 103.4° and crisis occurring as early as the third day.

(pseudo-crises). Finally, in the majority of cases of croupous pneumonia the temperature suddenly falls by crisis on the seventh to ninth day (Fig. 228) and convalescence is established, although the sudden fall of fever may be associated with dangerous collapse. Sometimes convalescence is broken by brief and slight febrile movements. If the case has been prolonged, or if it is of the typhoid type, the fever may end by lysis.

FIG. 228.

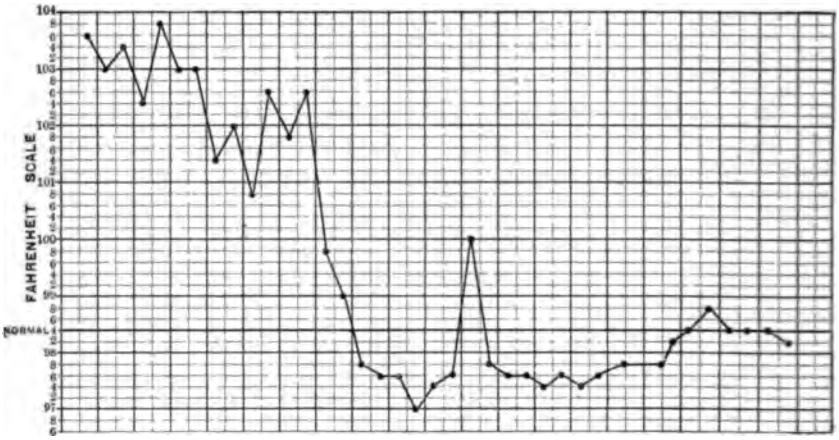


Chart of a case of croupous pneumonia, with crisis on the seventh day; admitted to author's wards on second day of illness.

It is to be remembered that the fever of *catarrhal pneumonia* is rarely as high as in the croupous form, usually 101° to 103°, and ends by lysis, not crisis. (See chapter on the Thorax.)

The fever of *acute bronchitis* possesses no peculiarities over that of other acute inflammations.

It is not proper to leave the subject of fever due to the various infectious diseases without calling attention to that due to ***syphilis*** in the secondary period of its course. With the onset of the roseola or other skin-lesion a fever, more or less marked, is nearly always present and is often preceded by chilly sensations and general malaise. This febrile movement may then follow one of three courses: it may never rise above 101°, and proceed as does a simple fever, with slight morning remissions and evening exacerbations; or it may be as remittent as is a malarial remittent fever; or, again, it may resemble a malarial intermittent, rising to a high point and then falling almost to the normal. Phillips, of London, has reported a case of syphilitic fever in which this febrile movement lasted for weeks, and, after being treated by quinine as a supposed tertian ague, ended at once under antisyphilitic medication. (See chapter on Skin, Eruptions.)

In *anthrax* (splenic fever) the temperature rises rapidly and becomes very high, and in the course of from three to five days becomes subnormal, when death occurs. The history of exposure to possibly infected hides or hair, the early development of a papule, vesicle, and pustule, surrounded by brawny swelling, and enlargement of the neighboring lymph-glands, render the diagnosis easy; but if any doubt exists, it can be promptly dispelled by a microscopical examination of the fluid from the pustule, when, if the disease be anthrax, the anthrax bacilli will be found. (See chapter on Skin.)

Fever with a vesicular eruption about the lips and on the mucous membrane of the mouth accompanied by disorder of the stomach and bowels may be due, particularly in children, to infection by the milk of cows suffering from *foot-and-mouth disease* or epidemic stomatitis. The prognosis is generally exceedingly unfavorable.

In *cholera Asiatica* during the stage of collapse the surface is very cold, but the rectal temperature may be found as high as 103° or 104°.

In *cholera infantum*, which is a form of gastro-intestinal irritation often produced by infected milk, there may be fever amounting to 102° or 103°, and not uncommonly much higher, even to 107° or 108° in fatal cases. The diarrhœa and obstinate vomiting, the age of the patient, the season of the year (usually hot weather), and the profound wasting, all complete the array of facts necessary for diagnosis. It is important to remember in these cases that the skin may feel cold and clammy even when the rectal temperature is very high.

The febrile movement associated with the progress of *acute parenchymatous nephritis* may or may not be preceded by a chill. The temperature may rise to from 100° to 104°, but the course of the fever itself is of no diagnostic import. The pulse, pain in the back, headache, perhaps drowsiness and coma, and the diminished urinary secretion, bloody urine, and albuminuria, render the diagnosis easy and the cause of the fever evident.

Very marked fever up to 104° or 105° may develop in the early course of acute infectious tonsillitis or in suppurative tonsillitis.

The fever seen in most cases of *tetanus* is very moderate, but it is subject to excessive fluctuations, and in cases approaching a fatal ending may reach 110°.

The fever occurring in *acute appendicitis* is a very unreliable symptom, notwithstanding assertions to the contrary. It rarely rises above 101° or 103° and sometimes not above 100°. Even in those cases in which the peritoneum has become involved by the inflammation the fever may not be marked, particularly if the peritonitis is septic. In other words, the presence of fever in association with pain in the right iliac region is a positive sign of some irritative or inflammatory process; but if the physician excludes appendicitis on the ground that fever is not present, he may make a serious mistake.

The fever of ordinary cases of *acute articular rheumatism* is usually moderate, rarely exceeds 103°, and possesses no typical characteristics; but in very severe forms of the disease with cerebral manifestations, a rheumatic hyperpyrexia may be developed, when, with delirium, convulsions, and cyanosis, the fever rises to 106° and even to 108°, after which death often ensues. The history of previous attacks of articular rheumatism, the hot, swollen joint or joints (usually the large ones), and the successive invasion of other joints as the ones first affected get well, point to the correct diagnosis. It must not be forgotten, however, that gonorrhœal and other forms of septic arthritis occur with febrile movement. Pyæmia, osteomyelitis, and purpura also may produce a fever with swelling of the joints. (See chapter on Legs and Feet.)

When a person, previously afebrile, during hot weather or when exposed to artificial heat in excess, is attacked by unconsciousness, convulsions, and very high fever, he is probably suffering from *thermic fever* or heat-stroke. Theoretically similar symptoms might be caused by a lesion due to embolism or hemorrhage in the neighborhood of the pons Varolii, but this is very rare. (See chapter on Monoplegia and Face and Head.) The fever in sunstroke may rise as high as 110° or 112° or even more ; the skin is hot and dry, or more rarely cold and moist with sweat; but, even if this is the case, the rectal temperature will be found hyperpyretic.

A great rise of temperature (110° to 112°) often occurs after injuries to the cervical region of the spinal cord.

Fever of considerable degree may be met with in cases suffering from hysteria, acute febrile neuritis, infantile spinal paralysis, apoplexy, and acute myelitis.

Temperatures as high as 106° have been reported as occurring in *hysteria*, but in a certain number of cases these records are really fictitious and produced by some trick with the thermometer. Only the rectal temperature, taken while the physician is present, should be relied upon in such cases.

The rapid development of fever, pain in the back and limbs, and particularly in the nerve-trunks, the temperature soon reaching 103° or 104°, may be due to an attack of *acute multiple neuritis*, and the history that the illness has followed exposure to cold and wet may, on the one hand, make the physician believe that his case is suffering from rheumatism or influenza, or on the other, in the absence of such a history, from the early stages of one of the infectious diseases. The early appearance of tingling, numbness, loss of power, and wasting of the muscles soon decides the diagnosis in favor of neuritis. The nervous disease which most closely resembles acute febrile neuritis is Landry's paralysis, and a differential diagnosis may be difficult; but in neuritis there are loss of sensation, muscular wasting, signs of degeneration, and fever, whereas in Landry's paralysis all these are wanting, excepting the sensory symptoms, which in both diseases may be similar. The predominant symptoms of Landry's paralysis are paralysis and loss of reflexes. (See chapter on the Legs and Feet.)

The prognosis of the severe form of febrile neuritis is grave, as death may ensue from respiratory paralysis.

The fever of *infantile spinal paralysis* (anterior poliomyelitis) often is the chief symptom ushering in the disease, and rises to 104° or even 105° in some cases. There may be convulsions, headache, and twitching of the muscles, and, after the acute attack has passed off, loss of power is speedily discovered in several muscles of one limb, as a rule. (See chapter on Legs and Feet.)

Fever at first amounting to only one or two degrees, but afterward rising as high as 104°, associated with numbness and weakness of the legs and loss of reflexes, followed by paraplegia, may be present as a symptom of *acute myelitis*, traumatic or otherwise. (See chapter on Legs and Feet, part on Paraplegia.)

A rise of several degrees of fever may come on after an epileptic convulsion.

A febrile attack not rarely seen in children, yet not readily placed under the heading of any given disease, has been described by Donkin and Goodhart, and the writer has also met with it quite frequently. This condition has been called *gastro-pulmonary fever;* but as either pulmonary or gastric signs may be absent, this term does not apply to all cases. A previously healthy child is suddenly seized with marked fever, rapid respirations, and râles may be heard in its chest. There are often vomiting, headache, and drowsiness, with recovery taking place in several days. Often these attacks are associated with gastric catarrh, but sometimes this state is not present. Donkin states, and it is the writer's own experience, that they are apt to be produced by fright or excitement. In a case of the writer's the sight of an angry skunk attacking a pet dog produced a violent attack.

Subnormal temperature of the body is seen as the result of any profound nervous shock, as after an accident or surgical operation, or prolonged anæsthetization. It occurs, too, at the ending of the fever of croupous pneumonia and other febrile movements ending by crisis. It is also seen in severe cholera morbus and cholera Asiatica and sometimes in cholera infantum, and often is present either in the early part of the cold stage of intermittent malarial attacks or more commonly after the fever of the attack has fallen. A subnormal temperature of a dangerous degree is met with in the algid type of pernicious malarial infection, and can only be satisfactorily differentiated from other conditions by a blood examination. Subnormal temperatures are also seen in some cases of confusional insanity and of tubercular meningitis and hysteria.

An important variety of subnormal temperature is that seen in the form of heat-stroke called heat-exhaustion, when, in place of fever, a condition of collapse is induced. Severe injury to the dorsal region of the spinal cord often produces a great fall of temperature. A temperature below 92.3° is nearly always fatal in its prognosis, but subnormal temperatures above this degree are not necessarily followed by death. A temperature of 95° is spoken of as one of moderate collapse.

CHAPTER II.

HEADACHE AND VERTIGO.

The causes of headache—Digestive headache—Headaches due to the eyes—Headaches due to cerebral growths and abscess—Headaches due to syphilis—Headaches complicating acute diseases.

HEADACHE is, of course, always a symptom and never a disease, and it arises from such widely different causes that it is impossible in this book to discuss all of them. Only the more common conditions resulting in its development can be considered, more particularly in relation to its diagnostic significance in serious pathological states. The most common cause of headache is probably disorder in the function of the digestive apparatus, the next most common cause is eye-strain in its various forms, and the third is nervous exhaustion or neurasthenia with or without associated anæmia. These may all be considered as perversions of function causing headache—that is, the pain in the head may be termed a functional headache. Less frequent, but far more important from a diagnostic stand-point, is headache seen in persons suffering from renal disease, brain-tumor, and meningitis in its various forms. The remaining causes of headache are numerous, and some of them will be considered later; but the most important of the first class are the headaches of the gouty or the rheumatic, and of the second class those of cranial periostitis, middle-ear disease, and acute inflammation of the eye or in the jaw.

Headaches depending upon disturbance *of the digestive system* are nearly always accompanied by evidences of such disorder, consisting in gastric or intestinal distress, belching, hiccoughing or vomiting, or even by diarrhœa. Often there is a distinct history of the ingestion of indigestible food or digestion-disturbing drink, but in other cases exposure to cold so congests the abdominal viscera that catarrh of the stomach and bowels is induced, and with it congestion of the liver followed by jaundice. The headache of disturbed digestion is nearly always frontal, and in many cases congestive to such an extent that the face may be flushed, or at least the intracranial circulation is so disturbed that the patient is unable to lower the head, because such a posture increases the pain. Such cases are relieved by hot foot-baths which relieve the congestion of the head; nearly always by the act of vomiting, which should be induced, if need be, by an emetic or by putting the finger into the back of the throat. Vomiting makes such headaches very much worse for a time, owing to the congestion of the head following the efforts at vomiting, and this is an important point in diagnosis, for in uræmia due to renal disease and in some other states the vomiting is often so easily performed that no straining accompanies it.

That disturbances of the digestive tube are capable of altering the intracranial circulation is proved by numerous facts. Thus Brunton quotes the experiments of Ludwig and Dogiel, who showed that moving the intestines by the finger introduced through an abdominal incision caused a great increase in the flow of blood through the carotid arteries.

Headache due to disorder of the digestion rarely ensues immediately after food is taken, since some time must elapse before the ingested material becomes changed into an irritating or toxic mass by fermentation or putrefactive processes. As a consequence, several hours or even a day may pass without any discomfort in the head, after which time the full force of the headache develops. The headaches of indigestion are, however, characterized by two important facts, viz., that they are not constant, and, second, that they are often relieved or prevented by the use of a purgative, even if constipation has not been present. Such headaches are very apt to be pulsating and accompanied by great nausea. Sometimes such a headache takes a form called migraine or hemicrania, a condition in which the pain is chiefly, if not entirely, unilateral, and there is associated with the pain early and more or less persistent hemianopsia. It is to be remembered, however, that in some cases of hemicrania of nervous origin the sickness at the stomach seems to be secondary to the severe pain in the head.

Headaches resulting from digestive disturbance do not always depend entirely upon irritation of the stomach and bowel with reflex disturbance of the circulation and sensory nerves of the head, but upon the absorption of poisonous substances formed in the digestive tube. These poisons are usually formed only to be destroyed by the liver, or are developed in too small quantities to have any effect; but no sooner do congestion of the liver and deficient biliary secretion ensue than they are formed in large amounts, and enter the general blood-stream, owing to the absence of antiseptic bile and the coincident or consequent constipation. As a result, we see very violent headache in jaundice due to catarrhal changes, particularly if the kidneys are not active in the elimination of toxic substances. Similar symptoms to those just described may occur in cases suffering from paroxysmal hæmoglobinuria, for in this state severe headache, nausea, vomiting, and persistent yawning are often present, with an icteroid discoloration of the skin. The reddish urine, pain in the liver, and sometimes an urticarial eruption will aid the diagnosis of this primary hæmoglobinuria.

In other cases in which no jaundice is present violent headaches, which utterly incapacitate the patient, come on from auto-intoxication. Thus, a man apparently perfectly well goes to bed on a certain night and wakes in the morning feeling a little more drowsy than usual. On rising he may feel a little stupid, and perhaps be slightly vertiginous, but is able to eat his breakfast as heartily as usual. In the course of a few hours the mental heaviness becomes more marked and a pain in the brow develops, which gradually gets worse and worse till it is unbearable. The ordinary remedies for neuralgic headache are futile, and he finds no relief until by the use of a purgative he removes the source of his intoxication, and his kidneys have time to eliminate the toxins already absorbed. Sometimes vomiting comes to his relief, and the emptying of the stomach so stimulates his liver and intestines by the efforts of vomiting that the process of auto-intoxication ceases. Some of the intestinal poisons have been isolated by Brieger, Harnack, and others, and have a physiological action like many well-known drugs. Thus, one produces effects like those of digitalis, another like those of belladonna, and a third like those of aconite. Pulsating pain and a slow, full pulse may indicate the absorption of the digitalis-like toxin; a flushed face and hot, dry skin, the belladonna-like toxin; and pallor, faintness, and a feeble pulse, if no nausea is present, the presence of the aconite-like toxin. Persons suffering from headache of this type are nearly always much freer from discomfort in the head after such an attack than they have been for some time before.

Brunton has also pointed out that digestive headaches are often associated with an objective and subjective sensation of increased intraocular tension and tenderness on the upper surface of the eyeball, and the author has frequently confirmed this observation.

The headache of *eye-strain* is usually due to abnormalities in the ocular muscles. Most commonly, according to Noyes, the externi (abductors) are the muscles which are the seat of the difficulty, but this opinion is not generally shared by other ophthalmologists, who assert that the interni are most commonly at fault. Such headaches may be felt in any part of the head, but are most commonly said to be in the occipital region. If, in association with such headache, immediately after or long after reading, there is blurred vision, pain in the muscles of the eye on suddenly moving the eyeball, any tendency to congestion of the lids, or hyperæmia in the conjunctiva over the insertion of the muscle, the diagnosis of headache from eye-strain is practically certain. (See chapter on Eye.) Violent pain in the head may also be due to irritable retina and to astigmatism and spasm of the ciliary muscle. Acute inflammatory processes in any part of the eye may produce severe headache, particularly iritis, the pain of which is very apt to be worse at night.

Whether muscular asthenopia or eye-strain can cause "sick headache" by reflex irritation is still undecided, but those ophthalmologists who are inclined to the theory of the influence of refraction errors in the production of morbid symptoms believe that it can.

Violent headache is often produced by *acute* or *chronic glaucoma*, and is usually felt about the eyes or orbit. Often it is of a unilateral character, and the sharp, shooting pain causes a false diagnosis of neuralgia to be made, or in some cases the patient is thought to be suffering from migraine, because in addition to unilateral pain there are often nausea, vomiting, and pallor of the face. The examination of the eye will show glaucoma to be present. Quite similar symptoms may appear as the result of a foreign body lodged in the cornea.

The headache associated with *nervous exhaustion* or neurasthenia may be superficial or deep; that is to say, neuralgic or apparently within the skull. It is often associated with some dizziness and vertigo, and is nearly always occipital in character, more rarely appearing over the brows. In addition to the pain, which is generally not very severe, there is often a sense of constriction about the head. Such a headache persists as long as a person who is overworked persists in fatiguing himself, and rapidly disappears when rest is taken. More rarely the pain in the head in neurasthenia is that of migraine, and is complicated by hemianopsia and hemicrania, often by a dilated pupil on the affected side, and flushing and pallor of one side of the face.

Headaches due to *rheumatism* are often quite severe, and are associated with much tenderness of the scalp or muscles covering the skull. Similar headaches, but more dull in character, are also seen in persons suffering from phosphaturia, and are relieved by benzoate of ammonium.

A headache is a symptom very commonly seen in persons who are subject to the chloral-habit, and it may be general or limited to the forehead. It is commonly associated with vertigo, flushing of the face, and intense heaviness and drowsiness.

Headache of a violent, bursting character may be produced by full doses of nitroglycerin, the salicylates, and quinine, and by the use of large quantities of tobacco.

Leaving the headaches due to functional disturbances not associated with organic change, we pass to those due to organic disease. Those due to *renal disease* are of two classes, in that they are an evidence of uræmia, or they are congestive and due to the high arterial tension so often seen as the result of chronic contracted kidney with its associated conditions of cardiac hypertrophy and arterio-sclerosis. Uræmic headache, as pointed out in the chapter on Vomiting, is often associated with nausea or vomiting of a persistent type, and sometimes with diarrhœa, for purging is an effort at elimination. The pain is not of the shooting, darting, or neuralgic type, but dull, even if severe, and is often associated with a sensation of fulness in the head. Sometimes the tendency to drowsiness is very marked, and, even if the patient does not sleep, he may seem on the verge of sleep all the time. Not rarely these cases instead of becoming comatose become wildly delirious.

These uræmic headaches may occur in any form of renal disease, acute or chronic, which results in uræmia; but, if the cause be chronic contracted kidney, there will be a high arterial pressure, and often a strongly beating heart with an accentuated second sound. This form with high arterial pressure will often be relieved by full doses of nitroglycerin, which not only relieves the tension, but also produces an increased renal activity. The urinary examination is of the utmost importance, and no surely correct diagnosis can be made in any case of suspected kidney trouble till this secretion has been examined and found abnormal. (See chapter on Urine.)

While headache is far less common as a symptom of *diabetes* than of nephritis, it occurs in the former disease either as a dull pain with lassitude and depression of spirits or as violent neuralgia.

Headache which is constant, although it usually varies in degree, may be due to *brain tumor*, and is one of the most important symptoms to be noted in the diagnosis of a case in which such a lesion is suspected. The pain is often worse at night, and is usually more severe in persons suffering from tumor of the cerebellum than in cases in which the growth is in the cerebrum, probably because cerebellar growths often cause effusion which produces pressure inside the skull. A tumor of the cerebral cortex, as a rule, produces more pain than one in the white matter beneath. Meningeal growths are also apt to produce severe headache, but bony tumors of the skull often press upon the brain to an extraordinary degree without causing any symptoms.

Headaches due to brain-tumor often have exacerbations with a regularity suggesting malarial disease, and, conversely, care should be taken not to mistake malarial headache for brain-tumor.

After constant headache, the most valuable confirmatory evidence of brain-tumor is papillitis of the optic nerve, which is present in about 80 per cent. of the cases. There may also be vomiting, and convulsions if the growth be in the motor cortex. Local paralysis, indicating the position of the growth, may be entirely absent, or it may exist and yet utterly mislead the physician as to the focal area which is diseased, since cases are on record in which, for example, a hemiplegia has existed, and at the post-mortem examination the growth has been found in the frontal lobes. Tumors of the base of the brain cause focal symptoms most commonly, and in addition to unilateral choked disk we find in many such cases ptosis from paralysis of the oculomotor nerve, disturbances in the functions of the trifacial nerve in its sensory filaments, so that painful tic (see chapter on Face and Head) or anæsthesia of the face may be present, and complete facial (Bell's) palsy may occur. If the hypoglossal nerve is affected by the pressure, the tongue is protruded to one side, it develops hemiatrophy, and disorders of speech result. Hirt points out that a tumor in the anterior fossa is apt to produce paralysis of the olfactory and oculomotor nerves and the upper branch of the trifacial. A tumor in the pituitary body causes pressure on the chiasm with resulting amaurosis, ptosis from oculomotor palsy, internal squint from paralysis of the abducens (sixth), and anæsthesia of the skin and muscles of the eyebrow, forehead, nose, and eye, from involvement of the first division of the trifacial. A tumor of the middle fossa above the dura causes oculomotor palsy (ptosis), patheticus paralysis (downward deviation of eyeball from paralysis of the superior oblique), and amaurosis from pressure on the chiasm. On the other hand, if it is below the dura, the oculomotor, the pathetic, the abducens, and the fifth nerve are paralyzed. When tumors occur in the posterior fossa they cause paralysis of the trifacial, facial, auditory, glosso-pharyngeal, vagus, spinal accessory, and abducens, or, in other words, cause anæsthesia of the upper part of the face, facial paralysis, deafness, loss of taste, irregular cardiac action, loss of power in the sterno-mastoid and trapezius muscles, and internal squint. Tumors of the lenticular and caudate nucleus, the interior portion of the thalamus, the corpus callosum, the fornix, choroid plexus, and of any part of the cerebellum except the vermiform process, may be present without any localizing signs.

Still more localizing symptoms are early paralysis of the oculomotor nerve from a lesion in the crus, hemianopsia in tumor of the occipital lobe, and tonic convulsions with preservation of consciousness and a staggering gait in tumor of the vermis of the cerebellum.

Should amaurosis be present, very valuable data as to the position of the growth are to be had from a study of the functions of the eye. If the pupils react properly to light, this shows that the optic nerves and tracts are intact, or, in other words, that the ocular reflex arc is perfect, and that the lesion must be in the ocular centres further back. On the other hand, if the reflex is absent, the growth probably presses on the nerve or tract. (See chapter on the Eye.)

The failure of a pupillary reaction may, however, depend upon amaurosis from lateral hemianopsia, in which case we examine the patient for what is known as "Wernicke's sign of hemiopic pupillary inaction." This is done by throwing the light by the ophthalmoscope so that it falls upon the blind half of the retina. If the pupil does not react, we have in all probability a lesion of the optic tract of that side; whereas, if the pupil does react, we have evidence that the tract is intact, and there must be a bilateral lesion of the optic radiations of the occipital lobes, or in the centre of vision in the cortex. (See chapter on Eye.)

Other general symptoms of brain-tumor are slow breathing, particularly when the patient sleeps, a slow pulse, and, as the growth increases, symptoms of cerebral compression. It ought to be remembered that brain-tumor may be closely masked by the results of chronic nephritis, for in the latter disease we find headache, local palsies or spasms, and, more important than all, an optic papillitis, which used to be thought pathognomonic of brain-tumor. Albuminuria may be present in both diseases, but tube-casts can usually be found in renal disease and not in tumor. Both diseases may exist side by side.

Care should be taken in a case of constant and severe headache that it be not thought due to brain-tumor until the possibility of its being caused by a syphilitic arteritis, syphilitic meningitis, or syphilitic gumma, is excluded, for mental depression and crashing head pains occur in all of these states. This is the more important because arteritis is the most common result of syphilis, meningitis frequent, and gumma least frequent of all the cerebral complications of lues. The differentiation of gummatous tumor from cerebral tumor due to other causes may be impossible unless there be a history of specific infection or manifestations of syphilis in scars or other external signs of syphilis. Improvement in the symptoms under the use of iodides and mercury would indicate syphilis rather than a growth due to other causes. The presence of optic neuritis would indicate tumor or meningitis, and would exclude arteritis; and in tumor the pain is apt to be localized, while in arteritis and meningitis it may be diffuse. The chief symptoms of arteritis are those indicating failure of a proper blood-supply to the brain, as evidenced by giddiness, weakness of groups of muscles, difficulty in speech, so that words are dropped out, and, it may be, the presence of symptoms like general paresis. Paralysis, when it develops elsewhere than in the ocular muscles, in such cases is usually the result of arteritis, since the arteritis results in a thrombus; but when the ocular muscles are affected the lesion is probably due to meningitis or to nuclear lesions. This development of ocular palsy is of great diagnostic significance. (See chapter on Eye and Face and Head.) In meningitis, on the other hand, the symptoms are irritative, such as spasmodic paralysis and irritative fever.

The following differential diagnostic table aids in making a diagnosis; but it is to be remembered that all these conditions may be very obscure:

Syphilitic Arteritis.	*Syphilitic Meningitis.*	*Syphilitic Gumma.*
Headache diffuse, often absent; not severe. Not started by pressure on cranium.	Headache diffuse and rarely wanting; sometimes localized. Started by pressure or by percussion on head. Very severe.	Headache usually localized.
Hemiplegia or monoplegia frequent. Muscles affected are flaccid, and reflexes are absent. Paralysis often fleeting and limited to a few groups of muscles.	Paralysis, if present, associated with rigidity and contracture, involuntary spasms, exaggerated reflexes. Paralysis more widespread.	Distinct focal paralysis common. Paralysis associated with rigidity and spasm.

Syphilitic Arteritis.	*Syphilitic Meningitis.*	*Syphilitic Gumma.*
Optic papilla usually normal, sometimes syphilitic retinitis is present.	Optic retinitis with marked neuro-retinitis and abundant exudation along the vessels.	Choked disk often present.
Partial epilepsy rare.	Partial epilepsy common.	
Aphasia is transitory and intermittent.	Aphasia less complete but more permanent.	
Hallucinations rare.	Hallucinations common.	Hallucinations rare.
Pain in limbs rare and fleeting.	Severe pain in limbs of central origin.	
Intellectual functions feeble.	Intellectual functions not feeble, but may be drowsy.	
No active delirium.	Active delirium often present.	
Ocular symptoms rare.	Bitemporal hemianopsia due to compression of the chiasm. Homonymous hemianopsia. Amaurosis from pressure on optic tracts.	Ocular symptoms of gumma involve ocular cranial nerves (see text).
Disorders of sensation are fleeting.	Permanent zones of hyperæsthesia, anæsthesia, and paræsthesia.	
Paralysis of cranial nerves not common.	Paralysis of any cranial nerve.	Paralysis of cranial nerves if gumma is so placed as to injure them.
Temperature may be raised.	Temperature quite frequently raised.	Temperature very rarely raised.

In connection with this table it must be remembered that, should the arteritis result in degenerative changes descending the pyramidal tracts, or in thrombosis with degeneration, the flaccid paralysis characteristic of arteritis may become spastic. Again, should aneurism arise from the arteritis the pressure upon a cranial nerve may produce paralysis, as does meningitis. Then, too, the meningeal symptoms may be varied. If the lesion is acute and at the base, there will be vertigo, compression of the cranial nerves, polyuria, and bulbar phenomena, and finally fatal coma. If it be at the convexity, then noisy delirium, convulsions, hallucinations, and paralysis in the form of hemiplegia or monoplegia appear. Death comes in coma. If it is chronic meningitis of the base, then we may have slowly developing alternate hemiplegia, crossed paralysis of the face and body, anæsthesia of one side of the face, and paralysis of motion on the opposite side of the body. If the convexity be affected then great irritability of the intellect, sensation, and motion may be present. Paralytic strokes are common, but coma is rare.

Violent headache is the most marked symptom of *brain-abscess;* but focal symptoms—that is, localized palsy pointing to the area of the abscess—are very often absent, although the localizing symptoms which have just been described as due to tumor may, of course, be due to abscess if it is so placed as to press on nerve-tracts or centres.

The rises of temperature which frequently occur in cerebral abscess are also indicative of the presence of pus, while the more rapid course of the disease, often only one or two weeks, points to abscess rather than tumor. Further than this, choked disk is rare in abscess and common in cases of tumor.

The difficulty of separating the headache of brain-tumor from that due to brain-abscess is very great, for the symptoms with the headache are almost if not quite identical in both cases. One of the most important of the differential points is the history of an injury to the head or of the presence of an infecting focus which could have caused cerebral abscess. Another means of aiding diagnosis is to examine the blood for leucocytosis. If the white cells are excessive, abscess is probably the cause of the illness.

In some cases of acute cerebral abscess, particularly in children, there is a curious tendency to bore the head into the pillow, or, if the child is still about the room, the head is rubbed or butted into the wall or against the body of the nurse. These symptoms are, however, absent in the slow, insidious forms.

When the physician has made a diagnosis of cerebral abscess from the headache and associated symptoms, he must not be misled into a reversal of his diagnosis by marked improvement in the patient, who may so far recover as to go back to his occupation, for it sometimes happens that a remission or latent period develops in the subacute forms of abscess. During this apparent remission, however, the temperature is rarely constantly normal, the patient is anything but well, and chilly sensations may be present.

Severe headache well diffused over the skull, coming on rather rapidly and associated with fever, stiffness of the back of the neck, vomiting, photophobia, delirium, and finally, stupor and paralysis, is probably due to meningitis or to tubercular meningitis, effusion at the base of the brain, or, more rarely, to the onset of a severe attack of one of the acute infectious diseases. The differentiation of the former from the latter is sometimes difficult, but the finding of some local tuberculous focus, the insidious nature of the onset in some cases, the family history, and a set of symptoms pointing strongly to involvement of the base of the brain indicate that the bacillus tuberculosis is the cause of the disease. The fact that these symptoms are due in some cases to the onset of one of the acute exanthemata is established promptly by the appearance of a rash.

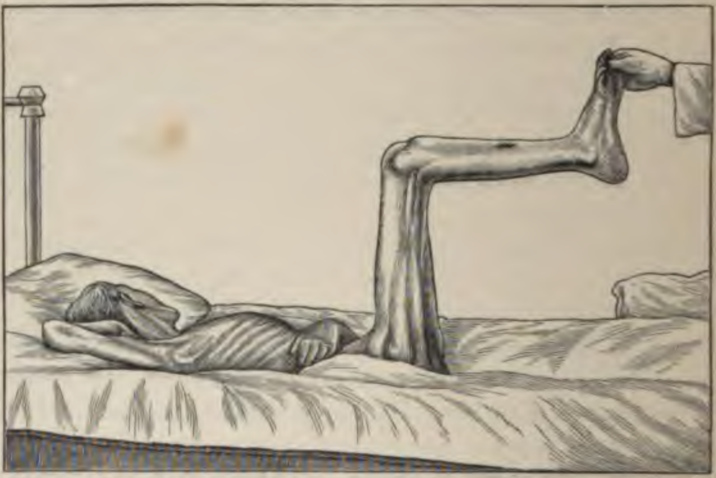
If the disease be tubercular meningitis, the head-pains will often be paroxysmal in character, so that the patient will at intervals of varying length give vent to sharp cries, evidently due to a sudden dart of pain. Vomiting may also be present and ocular symptoms develop, such as ptosis, strabismus, and unequal pupils, which have a sluggish reaction. The febrile movement will be irregular, now high, then very low; the temper peevish, if consciousness is present; and the skin pale and transparent. In the severe and rapid cases of tubercular meningitis marked delirium comes on, the patient picks the bedclothes, and there are tenderness and stiffness of the nape of the neck. Pulmonary signs of tuberculous disease are often present, and even if absent a focus of tubercular disease can often be found elsewhere. Care must be taken that the case is not mistaken for and thought to be typhoid fever, which it may closely resemble in its early stages, when headache, malaise, languor, and remitting delirium are present.

In children these symptoms of tubercular meningitis may be so marked as to lead the physician to the diagnosis of this disease almost at once. Usually for some two or three weeks before the onset of the severe symptoms the child will have been feverish and cross. Vomiting of a more or less obstinate form now comes on, and constipation is present. The pulse becomes slow and irregular, a mild fever is present, and emaciation may be rapid. The general nervous state is one of apathy, but finally may be disturbed by the sharp hydrocephalic cry. Often the child makes chewing or sucking movements. The fact, however, that several other conditions produce identical signs in this class of patients renders caution necessary. It has just been pointed out that the onset of an infectious disease may so result, and it is to be remembered that inflammation of the middle ear of an acute type may cause every one of the symptoms just described. Such cases are often incorrectly diagnosed until a discharge from the ear with great relief to the patient clears up all doubt as to the malady. Then, again, in some cases of croupous pneumonia all pulmonary symptoms may be masked in the violence of the meningeal manifestations, and, finally, it is not to be forgotten that in some cases of severe gastro-intestinal disorder there may be signs of meningeal inflammation, such as coma, squint, convulsions, myosis, Cheyne-Stokes breathing, and a depressed fontanelle.

In some cases of typhoid fever meningeal symptoms develop. In some of these the autopsy fails to show any signs of meningeal trouble. In others a true meningitis is found.

A valuable sign indicative of meningitis is that of Kernig. The patient is placed in a sitting posture at the edge of the bed, with the feet on the floor. If meningitis be present, it will be found that the leg cannot be extended on the thigh, because of contractures in the muscles. If the patient is too ill to sit up, then he should be placed on his back, the thigh flexed on the trunk and the leg on the thigh, and an attempt made to straighten the leg, which attempt will fail in a large number of cases if meningitis is really present.

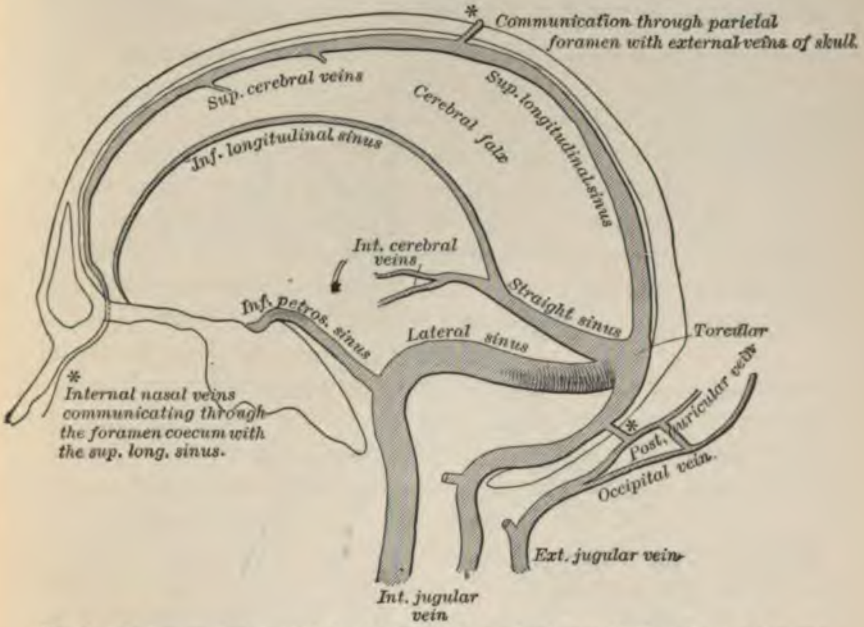
FIG. 229.



Kernig's sign, showing the strong contraction of the flexors on attempting to extend the leg. (After Osler's case.)

The symptoms of meningitis closely resemble those due to thrombosis of the cerebral sinuses, so closely, indeed, that only the presence of the typical signs of such occlusions can determine the diagnosis. Thus, if the superior longitudinal sinus is affected by thrombosis, there may be epistaxis from distention of the nasal veins, and the temporal veins will be swollen, and the near-by tissues œdematous through their close connections with the sinus through the emissary veins of Santorini, which escape from the skull by way of the parietal foramina. (Fig. 230.) In children there is usually in such cases bulging of the fontanelles and heaviness. Somnolence or delirium may be present with many of the characteristic symptoms of meningitis. This condition usually arises in connection with chronic exhausting diseases, such a long-continued diarrhœa and the continued fevers.

FIG. 230.

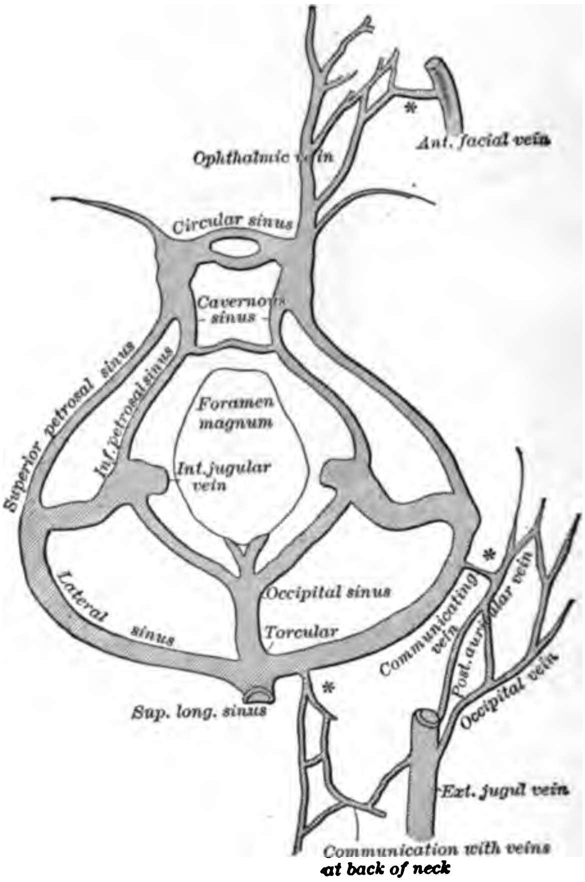


Showing the communications existing between the superior longitudinal and lateral sinuses and the external veins, indicated in the figure by *. (LEUBE.)

Thrombosis of the cavernous sinus is usually accompanied by quite typical symptoms. There is œdema of the eyelids and finally of the entire side of the face on the side of the affected sinus, but this facial symptom may be absent or very fleeting in its duration. Sometimes there is exophthalmos, and if the thrombus is septic a phlegmonous inflammation of the orbital connective tissue may occur. These symptoms are due to the communication between the sinus and the ophthalmic veins. Finally, as pointed out in the chapters on Face and Head and on the Eye, paralysis of the oculomotor nerve, the ophthalmic branch of the fifth nerve, and of the abducens and patheticus may occur, as these nerves pass through the cavernous sinus or in its walls. Nearly always thrombosis of the cavernous sinus results from some disease processes near by, as in disease of the middle ear and mastoid. Sometimes the affection is bilateral.

If the lateral sinus is affected by thrombosis, there is usually marked œdema back of the ear, owing to the clot extending to the small veins of the scalp, which pass through the mastoid and posterior condyloid foramina. The external jugular vein on the affected side is partly collapsed, particularly on full inspiration (Gerhardt's symptom). Rarely this vein may be unduly distended (Fig. 231).

FIG. 231.



Showing the communications existing between the lateral and cavernous sinuses and the external veins, indicated in the figure by *. (LEUBE.)

Thrombosis of the lateral sinus occurs far more frequently than that of the other sinuses. Suppurative otitis is its most common cause, and agonizing earache is, therefore, a symptom often associated with it.

Not only may cerebral thrombosis present symptoms resembling those of meningitis, but in addition those of cerebral abscess.

Violent headache, with vertigo, staggering, and confusion of thought, followed by unconsciousness, may follow meningeal hemorrhage due to disease of the bloodvessels, which are ruptured by some strain or by increased blood-pressure under the influence of stimulants. Hemiplegia or localized spasms may be present. The patient may survive several days in severe cases, or may recover if the hemorrhage is small; but usually a hemorrhage large enough to cause marked symptoms is large enough to cause death.

The individual affected by a meningeal hemorrhage will usually be plethoric, and, with the symptoms just described, will suffer from photophobia, extreme sensitiveness to the slightest noise, and pain radiating down the neck and trunk, which occurs in paroxysms. Localized paralysis is rarely present.

The presence of severe vertical headache in a middle-aged person who is insane and who is a male may indicate pachymeningitis interna hæmorrhagica (hæmatoma of the dura); but usually the insane patient does not complain, and an ante-mortem diagnosis of this state is not made.

Headache resulting from heat-stroke or thermic fever is usually the result of meningeal congestion or inflammation, and is one of the most annoying symptoms of convalescence. It is apt to be greatly increased by moving the head, and is often relieved by venesection.

The earlier stages of smallpox and pneumonia of the croupous type are often periods of violent headache, which symptom in the former instance decreases with the appearance of the rash, and in the case of croupous pneumonia so closely resembles the headache and associated symptoms of meningitis that a correct diagnosis, if the pulmonary signs are not sought for, may be impossible. In every case in which such symptoms occur the lungs should be examined.

When headache is present in the course of croupous pneumonia it often lasts till crisis, but in some cases ceases by the third day.

The chest should always be carefully examined in all cases of severe headache with fever for signs of pulmonary disease.

Headache is a constant symptom in many cases of typhoid fever in the early stages, but the peculiar tongue (see chapter on Tongue), the tendency to diarrhœa, the general systemic symptoms, and the facies of the patient will usually make its cause clear. More or less violent headache is often seen in measles, and depends probably to a great extent upon the engorgement of the nasal mucous membrane, or, in other words, has the same causative factor as has an acute "cold in the head" in producing cephalgalia.

Gruening is quoted by de Schweinitz as asserting that early morning headache is often due to nasal catarrh. This is, of course, only true if digestive troubles, which are often due to alcohol, and renal disorders are excluded. Severe morning headache, or dull headache on first waking up, may be due to nocturnal attacks of epilepsy, of which the patient is ignorant. If the tongue is bitten or the bed wet with urine, this diagnosis receives strong support.

Violent headache is often present during the febrile stage of intermittent fever and is often a complicating symptom of fever of the remittent type. In this connection the physician should remember that violent neuralgia of the supraorbital nerve is sometimes due to malarial poisoning, and is called "brow ague."

Headache is often due to anæmia, whether it be the result of hemorrhage or of the deficient formation of blood. The pain is usually frontal; there are often giddiness on movement, palpitation of the heart, a peculiar sensation in the head, and pallor of the skin. An examination of the blood will usually reveal the cause to be in this tissue.

Headache sometimes results from valvular heart disease. This in mitral regurgitation is often associated with vertigo, stupor, sleepiness, and, as night approaches, a mild delirium may come on. Its probable cause is congestion of the brain.

Rarely intracranial aneurisms produce headache, and when they are of the diffuse miliary variety this symptom may be a prodromal one before an attack of apoplexy. Large aneurisms may, however, exist without severe headache, and the position of the pain in no way indicates the seat of the aneurism, save that aneurism of the basilar artery may cause occipital pain. Auscultation might possibly reveal a murmur.

Headache may also arise from disease of the skull bones, either caries, osteitis, or periostitis, which result from injury, infection by syphilis or other infecting cause, such as typhoid fever or tuberculosis; but there is nothing diagnostic about the headache in these cases save that it is generally most severe in the area involved, and pressure over that part may elicit more or less pain or tenderness.

Violent neuralgia or shooting headache may be produced by exposure to cold, with resulting inflammation of the nerve-sheath; by dental caries, and by middle-ear disease or disease in the external auditory canal.

Vertigo.

Vertigo is a condition in which the patient feels as if he were losing his equilibrium. Sometimes he feels as if he were whirling around from right to left or left to right, sometimes as if falling forward or backward, and sometimes he seems stationary, while all his surroundings whirl round or rise up to or fall away from him. Although vertigo is a symptom which in itself lacks danger, it always produces great discomfort, if not fear. Functional vertigo arises from the patient being subjected to a whirling motion, from rough sea-voyages, and from indigestion, deficient circulation, or excessive cerebral congestion. Often it is due to cerebral anæmia arising from excessive hemorrhage. When it arises from indigestion it is probably due to reflex irritation, and perhaps to the absorption of toxic materials.

Vertigo as a symptom has a far more serious significance when it arises from organic disease. The most common lesions which cause it are middle-ear disease, Ménière's disease, tumors of the cerebellum, of the pons, of the crura cerebri, and the corpora quadrigemina. Vertigo also is not only a premonitory sign of an epileptic attack, but in the epileptic state called *petit mal* or minor epilepsy it is often the only symptom. In persons with atheromatous arteries it is very common, and sometimes it is a persistent symptom for some days before an apoplectic seizure. It is also present in disseminated sclerosis. Finally, many drugs, such as quinine and the salicylates, may produce it.

As the diagnostic points connected with most of the lesions here named are discussed elsewhere in this book, only Ménière's disease will be mentioned at this place. In addition to vertigo the characteristic symptoms of Ménière's disease are vomiting, noises in the ears, and, finally, deafness. The vertigo may be so severe that the patient falls to the ground. Aural examinations are usually futile in discovering any cause. Some authorities believe the disease to be due to a neurosis of the vasomotor nerves supplying the semicircular canals.

A form of vertigo unknown in America, the paralyzing vertigo of Switzerland, described by Gerlic, is a paroxysmal vertigo with great loss of power in the limbs, partial ptosis, and preserved consciousness.

CHAPTER III.

COMA OR UNCONSCIOUSNESS.

COMA is a condition of unconsciousness or insensibility from which the patient can be roused but partially or not at all, and it may arise from injuries to the head, while the patient is in otherwise perfect health, which injuries produce laceration of the brain-substance, cerebral or meningeal hemorrhage, or concussion. Again, it may be due to the influence of certain poisons, as alcohol, opium, chloral, cannabis indica, very large amounts of the bromides, or poisonous doses of other narcotics. Thirdly, it may arise from auto-intoxication, as in uræmia resulting from renal disease in cases of diabetes; in cases of profound exhausting disease, like typhoid fever or ulcerative endocarditis; or from acute yellow atrophy of the liver and pernicious malarial fever. Fourth, as a coincident symptom or sequel of hemorrhage into the brain (apoplexy), as the result of an epileptic attack, of a cerebral embolism or thrombosis, of thrombosis of the cerebral sinuses, of cerebral abscess, of pachymeningitis, leptomeningitis, or cerebro-spinal meningitis, of cerebral syphilis, of general paralysis, multiple sclerosis, and heat-stroke. The various points in connection with the diagnosis of coma from head-injuries are to be found in surgical treatises, and the history of a head-injury or the very presence of any injuries to the head is an important point to be sought after in the diagnosis. Care should be taken, however, to ascertain that any head-injuries found to be present are not the result of a fall due to the onset of sudden unconsciousness, rather than the cause of the coma.

The coma of *acute alcoholic poisoning* is characterized by profound insensibility, great muscular relaxation, loss of the ocular reflexes, and great fulness of the bloodvessels of the neck and face in the early stages, but finally by ghastly pallor of the face as the coma deepens on the approach of death. The skin is moist and warm at first, but afterward becomes cold. The pupils are usually moderately dilated; the pulse is rapid, at first strong, then more and more feeble, and the respiration stertorous and heavy. The sphincters, as a rule, are not relaxed, although they may be so in rare cases.

The bodily temperature in severe alcoholic poisoning progressively falls from 1° to 6° F. below normal.

Alcoholic coma is to be separated from that due to opium-poisoning by the absence of the contracted pupils and slow breathing of the latter condition, in addition to the other symptoms named below in discussing that condition; from coma due to cranial fracture by the absence of any history or sign of head-injury;[1] from chloral-poisoning by the history, the greater fall of body temperature, and the great feebleness of the heart and respiration produced by chloral. It may be impossible to separate alcoholic poisoning from that of cannabis indica-poisoning except for the fairly strong pulse generally found in the latter condition, and the history of the patient having taken the hemp or complained of the peculiar sense of prolongation of time before the coma came on.

The symptoms accompanying the coma of *opium-poisoning* are heavy sleep, preceding the deep unconsciousness, during which the patient can usually be aroused by shouting in his ear or by violent shaking, but sinks back into slumber at once on being undisturbed. The face is suffused and reddened and may be finally distinctly cyanotic, and the breathing puffing and stertorous. When the patient is awakened he breathes more rapidly, and for this reason the duskiness of the face disappears and the normal hue returns. Death never occurs in the second stage of opium-poisoning from the poison alone; but, if disease is present, death may take place at this time. The pupils are contracted to pin-points. The third or fatal stage emerges from the second by a gradual process, so that no abrupt line of separation can be noted. The face becomes at first more cyanotic, then pale and livid; the respirations, which have been eight to ten in the minute, are now only four or five; and, finally, such prolonged pauses occur that all hope of another respiration is lost by the attendant. While the slow breathing is at first deep, it now rapidly becomes shallow, and relaxation is present to the greatest degree. The skin, previously dry, is wet with the sweat of death; the patient is so deeply narcotized that nothing can arouse him, and he dies from respiratory failure, although the heart ceases almost simultaneously from the asphyxia. The pupils do not dilate in the third stage, except in the relaxation of death.

In view of the frequency with which alcoholic and opium-poisoning are confused, the following table is appended, which will be found of value in making a differential diagnosis as to the condition of the patient:

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[1] The physician must not forget that a fall from alcoholism may result in a cranial fracture.

OPIUM-POISONING AND ALCOHOLISM.

Opium-poisoning.	*Alcoholism.*
1. Pupils contracted.	1. Pupils normal or dilated.
2. Respiration and pulse slow and full.	2. Respiration nearly normal; pulse rapid, and finally feeble.
3. Face suffused and cyanosed.	3. Face may be pallid.
4. Skin warmer than in alcoholic poisoning.	4. Skin cool, perhaps moist.
5. Pulse slow, strong, and full till late in poisoning.	5. Pulse rapid, at first strong, then weak.

There is scarcely any difference as to consciousness in these two conditions.

When a *poisonous dose of chloral* is taken by man the person soon falls asleep and then sinks into a deep coma. The respirations become at first slow and labored, then shallow and feeble. The pulse, at first perhaps a little slowed, soon becomes rapid, thready, and shuttle-like, and is finally lost at the wrist. The face is white and livid, the forehead and the hands covered with a cold sweat, and the pupils, which are at first contracted, soon become widely dilated. Absolute muscular relaxation is present, and it is impossible to arouse the patient.

The coma of *uræmia* may come on gradually, but most commonly its onset is sudden and follows a uræmic convulsion. It possesses no diagnostic sign or signs which clearly separate it from the unconsciousness or coma following epileptic attacks, and, as the uræmic convulsion is often typically epileptic in character, the differential diagnosis is very difficult. An examination of the urine, if it can be obtained by the catheter or otherwise, will indicate, but not prove, the presence of uræmia if albumin be found in either large or small amounts, and the presence of very little urine in the bladder, indicating anuria, may be of some diagnostic significance. On the other hand, if the uræmia be due to chronic contracted kidney, the urine may be plentiful, the albumin scanty, but the low specific gravity be noteworthy. The pulse is usually very slow, often but forty to fifty, but the arterial tension is high, so that the artery feels hard and unyielding. The temperature of the body is usually very low in those severe cases which are free from convulsions and have a progressively downward course; so low a point as 91° to 95° being sometimes reached, at which time the patient is usually moribund from collapse. When convulsions are present the temperature may rise as high as 108°, and there may be in some cases a severe chill, followed by fever, and this again by collapse. The respiration is nearly always very deep, and sometimes very much quickened, and at times has a peculiar hissing sound. Sometimes the patient may purse his lips as if to whistle. Cheyne-Stokes breathing may be present. Rarely the breathing is difficult and asthmatic in type (uræmic asthma). If a preceding history of prolonged nausea, attacks of colliquative diarrhœa, and vertiginous symptoms can be discovered as having been present prior to unconsciousness, these will add to the array of uræmic probabilities. The coma of uræmia is not necessarily a fatal symptom. Even in very severe cases remarkable recoveries sometimes occur.

Coma resulting from *diabetes mellitus* is of far graver import, as it commonly terminates the patient's life. There may not be any prodromes, and there may not be any history of an exciting cause for the coma in a case of diabetes. Sometimes it is provoked in patients by severe exercise or great mental strain or emotion. When unconsciousness does not come on at once, the patient, after suffering from nausea, headache, and respiratory oppression, suddenly becomes anxious, delirious, and violent, then drowsy and deeply comatose. The pulse is not particularly noteworthy, but is usually full and not very tense. The respirations are deep and often very noisy, but at about the normal rate, although sometimes they may be rapid in the condition called diabetic dyspnœa. The body-temperature falls very greatly, even below 90° F. The respiratory changes and those in temperature may, therefore, be very much like those of uræmia; but in association with the coma of diabetes mellitus there are two pathognomonic symptoms: first, the sweet odor of the breath, which smells like the aroma of a pear or an apple, or a faint odor of chloroform; and, second, the presence of sugar in the urine, which secretion becomes dark red on the addition of chloride of iron. Testing for acetone usually reveals it in the urine in excess. (See chapter on Urine.)

Ordinary coma is rare in *typhoid fever*, and when it occurs is due to some complication, such as abscess or effusion at the base of the brain or meningitis; it is usually replaced by what is called coma-vigil, in which the patient, in a semi-conscious state, keeps muttering day or night. It is a grave sign.

The coma of *acute yellow atrophy of the liver* is generally preceded by headache, nausea, anorexia, and perhaps fever, followed by nervous excitement or restlessness, and then mental hebetude, which is often accompanied by a noisy delirium which may amount to mania. Finally, after several days, coma comes on and gradually becomes more and more profound till death takes place. Some of these symptoms resemble those of uræmia or diabetic poisoning, but the coma of acute yellow atrophy has in addition these characteristic signs, namely, jaundice, bile-stained urine, marked shrinking of the liver-dulness, enlargement of the spleen, and hemorrhages into the skin, or these effusions may take place into the bowels and stomach. The urine is singularly free from urea, but contains leucin and tyrosin in large amounts. (See chapter on Urine for description of leucin and tyrosin crystals.)

When coma comes on as the result of pernicious malarial infection, it is most apt to be ascribed to sunstroke, uræmia, or apoplexy, for its onset is usually sudden. Only a history of exposure to malarial influences, the presence of slight jaundice and anæmia, and of an enlarged spleen will serve to separate it from these conditions, and an examination of the blood for the malarial organism may be necessary before a positive differentiation can be made, for the diagnosis is by no means easy.

The coma of *apoplexy* may be sudden or gradual in its onset; generally it rapidly appears after the first symptoms of cerebral hemorrhage develop. The loss of consciousness may be partial or absolute, generally the latter if the leakage from a ruptured vessel be great. The respirations become stertorous, generally more rapid than normal, and, if a fatal result is in prospect, are rhythmically irregular; that is, they are now very slow, then gain in speed gradually till they become very fast, then the speed and vigor gradually fall till they are as feeble and slow as before (Cheyne-Stokes respiration). The history of preceding paralysis on one side of the body, or the presence of this loss of power if it can be demonstrated, the unequal pupils, the drawing of the face away from the paralyzed side, a strong, bounding pulse, and generally raised temperature complete the clinical picture of the coma of cerebral hemorrhage. If death does not ensue, consciousness may return, and the patient progress to recovery; but sometimes after several days of apparent convalescence a secondary fatal irritative coma comes on, associated with high fever. This is usually of ominous portent and is readily recognized because of the history. (See chapters on the Arms and on the Legs and on Hemiplegia.)

The coma of cerebral hemorrhage is unfortunately often taken for acute alcoholism, particularly as the latter state often produces the hemorrhage. The following table is designed to separate them:

ACUTE ALCOHOLISM AND APOPLEXY.

Alcoholism.	*Cerebral Hemorrhage.*
1. Pulse rapid, compressible, and weak.	1. Pulse apt to be strong and slow.
2. Skin moist, or relaxed and cool.	2. Skin hot or dry.
3. Bodily temperature lowered.	3. Bodily temperature raised.
4. Pupils equally contracted or dilated; generally dilated.	4. Pupils unequal.
5. No hemiplegia.	5. Hemiplegia; one side tossed, the other remaining motionless.
6. Breathing not so stertorous nor so one-sided in lips.	6. Respiration stertorous, the lips being inflated on one side on expiration.
7. No facial palsy.	7. Facial palsy.
8. Unconsciousness may not be complete.	8. Unconsciousness complete.

The smell of alcohol in the breath is no guide, as the high arterial pressure of acute alcoholism may have caused the rupture of a cerebral bloodvessel.

Coma due to *cerebral softening*, following embolism or thrombosis, has no signs other than those discussed in the diagnosis of these lesions in connection with hemiplegia (which see). Coma due to thrombosis of the sinuses of the brain is accompanied by the following diagnostic symptoms, namely, irritation or paralysis of the cranial nerves resulting in strabismus, nystagmus, and lockjaw, stiffness of the neck, and clonic spasms. If the cavernous sinus is thrombosed, there will generally be found stasis of the veins in the eye, which means retinal congestion. The eyeball may be protruded, the eyelids swollen, and perhaps loss of function in the oculomotor nerve may be present, causing ptosis, and, if the abducens is affected, causing internal strabismus from paralysis of the external rectus. If the transverse sinus is involved, there will probably be œdema behind the ear, and, if the petrosal sinus or internal jugular be obstructed, the proximal part of the vein collapses. Thrombosis of the superior longitudinal sinus causes epistaxis and engorgement of the temporal veins. Thrombosis of any of these sinuses, however, may be present without these signs.

Coma due to *subdural hemorrhage* (pachymeningitis interna hæmorrhagica) is peculiar in the fact that its onset is usually very slow, and the signs of nervous irritation last a long time and are quite violent, often amounting to epileptic paroxysms. Commonly, too, there will be rigidity of one limb, but the cranial nerves usually escape. The coma usually follows these signs, and the condition is peculiarly common in the chronic insane and in paretic dements.

Sudden unconsciousness with hemiplegia and vomiting may also come on in Raynaud's disease.

Coma from *cerebral abscess* is accompanied by symptoms closely resembling those of acute meningitis. The patient is dull and delirious; has headache, fever, and often has a hyperpyrexia. The sensibility becomes less and less, and deepens into the coma which ends in death if relief is not given. The localizing symptoms of paralysis may indicate that a lesion is in a certain part of the brain; but generally these signs are absent, because cerebral abscess is usually in the frontal lobes. If there is a history of injury, purulent otitis, infectious disease involving other parts, such as septicæmia from wounds or empyema, and if there are vertigo, vomiting and headache, fever, and an absence of choked disk of the optic nerve, the diagnosis is probably cerebral abscess; but a long duration of months is no sign that it is not abscess, as these cases often run a very prolonged course.

The coma of *purulent leptomeningitis* resembles that of abscess in many of its associated symptoms; but the intense headache, the rapid development of delirium and unconsciousness, the stiffness of the neck, the optic neuritis and disturbed movements of the ocular muscles, combined with the absence of a history of septic absorption, may make a differential diagnosis possible. Purulent leptomeningitis is rare, but it sometimes occurs in association with croupous pneumonia, and the presence of this disease will point to the cause of the coma. The coma due to *epidemic cerebro-spinal meningitis* is diagnosed by the characteristic rigidity of the neck, excessive headache preceding the unconsciousness, the disturbances of the cranial nerves producing strabismus, unilateral or bilateral ptosis, nystagmus, impaired pupillary reaction, mydriasis and myosis. The face is often painfully distorted. The presence of an epidemic, of course, makes the diagnosis clear.

It is well to remember that coma may be present from other forms of meningitis and arise in several conditions presenting similar symptoms, such as pneumonia of the meningeal type, otitic abscess, and gastro-enteritis. (See chapter on Headache and Vertigo.)

Cerebral syphilis may result in the development of coma by producing hemorrhage, embolism, arteritis, tumor of the brain, or almost any other lesion, and its diagnosis as the cause of an attack of coma is not easy. Of course, a history of syphilitic infection and the presence of symptoms of this condition in a patient who is too young to have secondary arterial changes from age render the probability of syphilis as a cause very great. Scars on the skin (see chapter on Skin) may show specific taint.

When coma results from *general paralysis* it usually succeeds the peculiar epileptic attacks which come on late in that disease, and the history of delusions, tremor of the hands, peculiar speech, loss of the reflexes, with earlier milder attacks, like the one before us, combined with the age of the patient, render a diagnosis possible.

Practically identical symptoms may attend the development of coma from *multiple sclerosis*, and without the history of the latter affection the diagnosis may be impossible. If this history shows a spastic gait and intention-tremor, nystagmus, mental weakness, and heightened reflexes, the probability of the attack being due to multiple sclerosis is increased.

Coma is sometimes seen as a later manifestation of *Addison's disease*, and it often develops very suddenly.

Heat-stroke produces coma as one of its almost constant symptoms. The history of exposure to heat and the hyperpyrexia are the two diagnostic points of importance. (See Fever.) Sudden unconsciousness may arise from *heart-failure* due to disease or fright; we call this fainting. Frequent attacks of this character should cause the physician to listen to the heart to discover if there is valvular disease, particularly aortic stenosis and fatty heart, and he should be on the outlook for renal difficulty. Sometimes sudden repeated attacks of unconsciousness are due to *petit mal* or minor epilepsy. Coma more or less profound follows attacks of true epilepsy. (See next chapter.)

CHAPTER IV.[1]

CONVULSIONS OR GENERAL SPASMS.

Definition of a convulsion—The convulsions of epilepsy in its various forms—Of infancy—Of hysteria—Tetanic convulsions—Tetany—Spasms—Chorea.

A CONVULSION is a condition in which by reason of sudden tonic or clonic contractions of groups of muscles the body in whole or in part is thrown into spasmodic movements. Convulsions can be divided into those which are clonic or epileptiform and those which are tonic or tetanic. Further, it is a general rule that convulsions which are epileptiform or clonic in character have their origin in the cerebral cortex, while those of the tetanic or rigid type arise from excitation of the motor tracts in the spinal cord. The clonic variety of convulsions are represented by idiopathic, traumatic, reflex, and syphilitic epilepsy, hysterical convulsions of an epileptic type, uræmic convulsions, and those convulsions which arise from the presence of growths or other sources of irritation in the cerebral cortex. Certain poisons may also rarely produce such attacks, notably lead and alcohol, and sometimes malingerers imitate very successfully the epileptic paroxysm.

The convulsion in **epilepsy** is characterized in some cases by the primary appearance of an aura—that is, a sensation in some part of the body, which the patient discovers comes on before each convulsion. This aura may be of any character and appear in any part. Most commonly it is sensory, and is as if a cloud or wave was passing up the body to the head. As the sensation reaches the head the patient may utter the peculiar epileptic cry or sigh, and with this sound the patient becomes rigid from tonic spasm of the muscles. this spasm now relaxes for an instant, and then the patient's muscles pass into a state of alternate relaxation and contraction which throws the patient's body from one place to another.

The primary tonic spasm of the face produces risus sardonicus in some cases: the head is often drawn to one side, the eyes commonly turned to the same side, and the lower jaw locked tightly against the upper jaw. The arms are strongly flexed at the elbows, the hands flexed at the wrists, and the fingers bent into the palms of the hands with great force. As a rule, the evidences of the powerful flexors overcoming the extensor muscles predominate; but sometimes the reverse is the case, and forcible, rigid extension of the parts affected takes place. The duration of these tonic contractions rarely exceeds two minutes, and in most cases is limited to but a few seconds.

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[1] For local spasms or tremors, see chapters dealing with Face and Head, Hands and Arms, Feet and Legs.

It is followed by clonic spasms, already described, which are ushered in by more or less violent tossings, but whose onset is forewarned by peculiar vibratory thrills which run through all the affected muscles. The eyelids tremble, the body changes its position never so slightly, and then, as if the vibrations gained greater and greater power with each moment, the fibrillary tremors give way to muscular contraction. The expression of the face, which in the preceding stage was set and firm, is now constantly changed by the movements of the facial muscles; the jaws, no longer locked together, are gnashed and crunched one upon the other; the tongue is alternately protruded and drawn back, and, as a consequence, is often caught between the teeth and lacerated. The excessive movements of the muscles of mastication force the increased quantities of liquid secreted by the salivary glands from the mouth in the form of froth, which is often stained with blood by reason of the injuries to the tongue. The constancy of the convulsive movements now becomes less and less marked; well-developed remissions occur between each toss of the body, until the movements cease entirely; but it should be constantly borne in mind that the prolongation of the remissions does not produce any decrease in the severity of the intervening spasm, the final spasm often being even more violent than the first.

The intense discoloration of the face begins to pass away as soon as the remissions, by their length, permit the blood to be oxygenated, its disappearance being temporarily arrested by each paroxysm. Finally, the spasms having ceased, the patient lies before us relaxed, unconscious, and exhausted, and usually passes into a deep sleep or coma, which lasts a variable length of time, and from which he cannot be aroused, except very rarely, and then with great difficulty.

When one part of the body is involved in an epileptic paroxysm, the rest of it escaping, the condition is called *Jacksonian epilepsy.*

By far the most important of its peculiar signs is the character of the onset, which always begins, in the typical Jacksonian form, in some peripheral portion of the body, and most frequently in the muscles of the thumb or hand, so that for the moment the convulsive movements are localized. They may remain localized at the point of origin, or immediately diffuse themselves over muscle after muscle until all the arm, leg, or other groups of muscles are involved. It is of the greatest importance, however, that the reader should keep the aura of an attack separate in his mind from the onset, remembering that the term onset is here used by the writer to designate the beginning of the period following the aura, if there be one. Jacksonian epilepsy may be of almost any severity. In rare cases only one muscle may suffer throughout an entire attack, but in others the entire body may be at last convulsed. There may or may not be loss of consciousness, its presence or absence being dependent upon the seat of the lesion in the brain and the severity of the attack. In those instances in which only a few localized muscles are involved consciousness is more commonly preserved than lost.

Typical Jacksonian epilepsy may develop in the course of general paresis.

An epileptiform convulsion may be associated with the onset of an apoplexy, and usually indicates that the hemorrhage is in the motor cortex. Such an attack is generally Jacksonian in character; that is to say, one muscle or a group of muscles is involved, or, if not this, the attack is, at most, only unilateral. The cause is made manifest by the presence of the symptoms of apoplexy as generally seen, for there are inequality of the pupils, drawing of the face to one side, and a consequent hemiplegia which lasts indefinitely. Of the attack itself, it may be said that, so far as the movements are concerned, they differ in no way from those of the true Jacksonian epileptic seizure; and it should be remembered that hemiplegia often follows ordinary idiopathic epilepsy. Such a post-epileptic hemiplegia is, however, usually fleeting, while that due to hemorrhage is more or less permanent. It should be remembered, however, that apoplexy may complicate epilepsy, being produced by the convulsion. Then again the lesions caused by a hemorrhage may ultimately result in epileptiform attacks, although this is certainly rare in adults. In some persons the history of this attack is very indistinct, owing to its occurrence in early life; while in others the paralysis has been so slight or temporary as not to bear any relation in the mind of the patient with the convulsive seizures following, which in many cases do not occur for some time after. The palsy and convulsions are not always due to hemorrhage, but to any pathological cerebral change. Heart disease, by causing embolism, may bring them on, and syphilis and puerperal sepsis may all produce a softening of the cortex, with an epileptic state following the paralysis.

We can very readily divide post-hemiplegic epilepsy into two classes, for we find that in about one-half of the cases the convulsion occurs along with the paralysis and then follows at intervals, while in the other half the paralysis is not followed by convulsive seizures for weeks, months, or years.

Post-hemiplegic epilepsy may occur at any age, but there can be no doubt that it far more commonly occurs in children than in adults. In at least two-thirds of the cases the onset is before five years of age, and in nearly one-half it is during the first two years of life.

The frequency with which post-hemiplegic epilepsy comes on in the hemiplegia of childhood has been very recently studied, and the conclusion reached that its occurrence is quite common. Thus, in Osler's cases twenty children out of ninety-seven suffered from it. In the eighty cases collected by Gaudard eleven children had hemiplegic epilepsy, and sixty-six children out of one hundred cases collected by Wallenberg were epileptic after hemiplegia. In another series of cases collected by Osler fifteen children out of twenty-three were thus affected. (See Cerebral Diplegia.)

Very interesting results are reached if the statistics of this condition are analyzed. Thus, it will be found that in the cases which date from infancy females are twice as numerous as males, but in cases after five years of age there is no difference between the frequency in the two sexes. One of the theories of these infantile cases has been that they were produced by the use of instruments during labor, and repeated post-mortem examinations have confirmed the possibility of this occurrence. On the other hand, every obstetrician knows that the birth of a boy generally means a more difficult labor than that of a girl, owing to the greater size of the head in a male child. *A priori* reasoning would seem to show, therefore, that the heads of male children would, accordingly, have instruments applied most frequently, and consequently that infantile cerebral trouble would be the result more commonly in males than in females;

but, as has been said, this conclusion is contraindicated by the facts. Another fact of great interest is that the paralysis in the infantile cases is more frequently on the left side than the right, but after the fifth year it is equally common on both sides.

The writer has already spoken of the fact that the convulsions may occur along with the first attack of paralysis, and continue, or that an interval may occur between the attack and the subsequent paroxysm. The chronic recurrent fits date from the onset in about one-third of the cases, but it is not uncommon for the paralysis to occur in infancy and the epilepsy to begin at puberty. It would seem that cells injured in early life may lie undisturbed till the increased demands of maturity call them out into diseased action. This prolonged interval occurring so commonly in children separates them from adults in this disease, for in the latter class it is very rare for the epilepsy to be delayed for more than one year.

Syphilitic epilepsy is only one of the many nervous affections which afflict those who may be so unfortunate as to contract this disease. There can be no doubt that syphilis produces epilepsy in adults.

There is also one symptom which may occur early in syphilitic epilepsy, or sometimes only late in the disease, namely, repeated partial, passing palsies, which while they may be in some cases hysterical, are in the syphilitic almost pathognomonic of brain-involvement —a momentary weakness in one arm; a slight drawing of the face to one side, which disappears in a few hours; a temporary dragging of the toe; a partial aphasia which appears and disappears; a squint which to-morrow leaves no trace behind it. (See Syphilitic arteritis.)

It is important to determine whether *idiopathic epilepsy* can be separated from that due to syphilis simply by the symptoms. This is very difficult to decide. So far as the convulsion itself is concerned, it is not possible to separate them. If, however, we can obtain any history, the matter becomes much more simple.

Fournier, in his lectures on epilepsy, in the Louvain, in Paris, in 1875, gave a summary of his views as follows:

1. In the *syphilitic epilepsy* there is nearly always absence of the shrill cry at the onset, so characteristic of the idiopathic variety.
2. There is frequently paralysis immediately after the attacks.
3. The seizure is incomplete or unilateral in character.
4. Attacks constantly increase in severity.

A therapeutic point, which may be used with the greatest success, is the administration of iodide of potassium in large doses. If the epilepsy be syphilitic, it will usually become less severe, and enormous amounts of the drug may be borne with impunity. As much as 450 grains in twenty-four hours will often do good.

The convulsions of an eruptive fever differ from the true epileptic attack very slightly indeed. It is only by the history of the patient and by waiting for developments that we can determine which is which, for as soon as the eruption or high temperature of an exanthem occurs the character of the attack is evident.

Epileptic convulsive disorders may arise owing to the action of a very large number of toxic substances, of which the writer shall here consider only a few, as an enumeration of all of them is manifestly impossible.

Alcoholic epilepsy consists of two distinct varieties produced by over-indulgence in intoxicating drinks. In one of these the convulsions are symptomatic of acute poisoning, and come on during a drunken orgy or immediately after a single large draught of liquor.

In the second variety the convulsion does not originate while there is alcohol in the blood, but in the intervals between the attacks of delirium tremens resulting from chronic excessive alcoholic indulgence. Under these circumstances the paroxysms are generally accompanied by hallucinations or by dementia or imbecility. In the alcoholic convulsion the symptoms may closely resemble those of true epilepsy, and not rarely the attack is ushered in by headache, gastric embarrassment, disorders of vision, and excessive tremors or some similar prodrome which may be looked upon as partaking of the nature of an aura. As a general rule, these alcoholic convulsions occur in paroxysms—two, three, four, or more, one after the other, at intervals of a few minutes. Not only may *grand mal* be closely simulated by alcoholic epilepsy, but simple vertigo or true *petit mal* may exist, either alone or associated with major convulsions. Alcoholic epilepsy is often associated with hallucinations, especially of terror, and not rarely is followed for days by a certain degree of mental disturbance. Rather curiously these cerebral disturbances result rather in suicidal than homicidal tendencies, which is just the reverse of the insanity following simple epilepsy.

The symptoms of an uræmic convulsion will be spoken of further when studying its differential diagnosis in connection with epilepsy.

The diagnosis of *lead epilepsy* from the idiopathic varieties is somewhat difficult, if the patient is seen for the first time during an attack; but the ordinary methods of determining chronic lead-poisoning are, of course, of equal value here. The blue line on the gums may be present, and, if so, the diagnosis is almost certainly lead-poisoning; but its absence is no proof that lead is not present. The administration of iodide of potassium also will so increase the elimination of the poison as to benefit the case and render it more easy to recover lead from the urine.

The history of exposure to lead in any form is, of course, exceedingly valuable evidence, but it should not be forgotten that in many cases this history is wanting. Thus, the poison may be derived from a hair-dye, or cosmetic, or from water which contains lead from pipes, or from an endless line of similar hidden and obscure sources. Amaurosis may be present in some cases, or optic neuritis with atrophy may occur. Where double wrist-drop is present the diagnosis is much more easy.

The symptoms of epilepsy due to chronic poisoning by lead are chiefly as follows: the man, apparently in his usual health, or who has had for a few days a feeling of weight in the head, or headache, is suddenly seized with most violent convulsions, which are often fatal, and which during their presence resemble ordinary epilepsy so closely as not to be separated from it. They end in coma, and are separated from each other by intervals of nervousness and disquiet. In some cases one convulsion follows the other so rapidly that death ensues from exhaustion, but in much more rare instances the attacks may resemble Jacksonian epilepsy very closely, and there may be no loss of consciousness. If such a condition occur, it is almost sure to be followed by a more violent fit. The attacks are not preceded by any aura whatever, but previous to the headache, already mentioned, the patient may have had amaurosis, and ophthalmoscopic examination of the eyes may show choked disk and neuritis of the optic nerve. As a general rule, such cases are fatal, but they may recover under careful treatment.

It is exceedingly important to differentiate between those convulsions which arise from uræmia brought on secondarily by an action of lead on the kidneys and those which are due to a direct action on the brain. This may be difficult from the mere symptoms presented, but there are some points of difference. In the first place, the convulsion of uræmia is, as a general rule, not so violent in its movements nor so sudden in its onset. It is generally preceded by a few days of somnolence, or weeks of gastric disorder and headache, while lead epilepsy is generally sudden or preceded by cephalalgia by only a few days or hours. Again, examination of the urine in uræmic convulsions will show a decreased amount of urea in proportion to the quantity of urine passed, while in plumbic epilepsy just the reverse will be true, unless the kidneys are affected *pari passu* with the cerebrum. If albumin be present, uræmia is pointed to; but if the urine has a slow specific gravity and is passed in large amounts the indications are that there is chronic contracted kidney, which may or may not be the cause of the nervous disturbance.

Malarial epilepsy is an uncommon disorder, even in countries and regions which are notoriously malarial, but it has probably occurred, particularly in the southern part of the United States and in Brazil. The only cases which the writer can find recorded are by American or English observers, namely, Jacobi, Payne, and Hamilton. The latter gives but a passing glance at the subject, and the articles of the others the author has not been able to obtain, so that he knows them solely by reputation. In Hamilton's case a young man, who had lived for many years in an exceedingly malarious region, had more or less periodic epileptic attacks, attended by great preliminary rise of temperature and intense congestion of the face and head. He was unusually somnolent, and in the intervals frequently suffered from facial neuralgia. Change of the place of habitation and the use of quinine removed the disease entirely.

The differential diagnosis between idiopathic epilepsy, that which is due to demonstrable cause, and the diseases which resemble it, is quite possible in many cases.

Undoubtedly the most similar convulsive condition that we have is that known as hysteria, and the diagnosis of one from the other is as difficult in some cases as it is essential and necessary for treatment and cure. The other conditions with which it might be confused are uræmia, alcoholic epilepsy, tetanus, and syncope. Below are arranged all these disorders in a table, which briefly and succinctly shows the different points between them, although, of necessity, it is somewhat arbitrary on account of the lack of space. Nevertheless, it is hoped that it will be clear enough to be of service.

TABLE OF DIFFERENTIAL DIAGNOSIS OF EPILEPSY FROM HYSTERIA,[1] ETC.

Signs.	Epilepsy.	Hysteria.	Uræmia.	Petit mal.	Alcoholic epilepsy.	Tetanus.	Syncope.
Apparent cause.	None.	Emotion.	None.	None.	None.	None.	Mental shock.
Aura or prodromata.	Generally present, but short.	Globus hystericus; palpitat'n: choking.	Headache, vomiting, and dyspepsia.	Faintness and dimness of vision.	Tremors.	Nervousness.	Not so well defined as in epilepsy.
Onset.	Sudden.	Often gradual.	Often gradual.	Sudden.	Sudden or gradual.	Gradual; begins in jaw.	Sudden or gradual.
Scream.	At onset and sudden.	During attack.	Frequently none.	Frequently none.	May or may not be present.	None.	None.
Convulsion.	First tonic, then clonic.	Rigidity more pronounced, with more aching.	Rigidity generally absent.	No rigidity.	Movements more clonic than tonic.	Always tonic.	None.
Biting.	Tongue.	People, tongue, lips, and hands.	Tongue.	None.	Rarely.	None.	None.
Micturition.	Frequent.	Never.	Never.	Rarely, except when bladder is affected	Rarely.	Sometimes.	Never.
Defecation.	Occasionally.	Never.	Never.	Never.	Rarely.	Rarely.	Never.
Talking.	Never.	Frequent.	Muttering.	Never.	Never.	Never.	None.
Duration.	A few minutes.	Generally many minutes.	From a minute to hours.	Momentary.	May be prolonged.	Hours.	Indefinite time.
Consciousness.	Lost.	Generally preserved.	Lost.	Not lost always, but clouded.	Lost.	Preserved.	Lost.
Termination.	Spontaneous.	May be induced by shock.	Spontaneous.	Spontaneous.	Spontaneous.	Spontaneous.	Gradual, with no somnolence.

As already stated, in epilepsy the movements are typically at variance with those of daily life, while in hysteria they are almost equally typical of ordinary muscular contractions, or, in other words, are more purposive in character, and frequently there is prolonged tonic contraction of the muscles, giving rise to the assumption of positions which bear more or less resemblance to normal attitudes. In hysteria, also, consciousness is impaired sometimes, but never so completely as in true epilepsy. Indeed, most commonly the individual knows all that goes on around her, for, while she may give no sign of consciousness by words or looks during the attack, she may afterward be able to narrate all that has occurred. Less commonly in hysteria, a condition known as automatic consciousness exists, in which, during the paroxysm, the patient understands all that is said, but forgets everything on the return to quietness.

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[1] This table is taken from the author's essay on Epilepsy, the prize essay of the Royal Academy of Medicine in Belgium, January, 1889.

The fact that the patient is a female cannot be regarded as affirmative evidence of hysteria in the least, but if convulsions occur in a male they may be taken as fairly positive evidence of the disease being epilepsy; and yet it should always be remembered that males may suffer from hysterical attacks.

The movements of the hysterical patient after the tonic condition has passed away are as clonic as those of the epileptic, but still possess some purposive characteristics, and are not so bizarre as are those of the true disease. Thus the head, arms, and legs are struck with evident endeavor against the floor or surrounding furniture. Another point, which, when it occurs, is very distinctive, is the onset, toward the close of an hysterical convulsion, of a second stage of tonic spasm, such as occurred at the beginning. It will be remembered that this does not occur in epilepsy, although it must be borne in mind that in cases of the "status epilepticus" the rapid onset of another attack may show a second tonic stage. This can be separated, however, by the fact that it is followed by clonic movements, whereas the secondary tonic stage of hysteria is usually followed by relaxation and temporary recovery.

In the secondary hysterical tonic contractions emprosthotonus and opisthotonus may occur, and are even more rigid in their character than they are in the first attack in some cases. Finally, too, in hysteria some peculiar emotional position is often assumed, as of the crucifix or of intense grief, or, perhaps, immoderate laughter is indulged in, with corresponding movements of the trunk. If the patient is quiet at this time, a smile may float across the face, while the eyes, with a look of pleasure, pain, or entreaty, may seem to be gazing at some object very far off. In some very well developed cases the expression of pleasure is followed by a look of pain, with painful movements, or an appearance of intense voluptuous entreaty, with sensual venereal desire evidenced by gestures. Great terror may be present, and, as the scene constantly changes, the woman is now joyous, now mournful, now scolding, now praising her attendants or herself. Such is the history of a fully developed attack of hysteria.

Hysterical convulsions in their fully developed form are comparatively rarely seen among Americans, Germans, Belgians, or corresponding races, but are very frequently observed by French practitioners of medicine.

In France there can be no doubt that the tongue is commonly bitten in hysterical convulsions, and that frothing of the mouth is frequently present; but in other countries this symptom may be regarded as indicative of epilepsy rather than hysteria. Doubtless the inexperienced reader will say, upon comparing these symptoms with those which were given as occurring in epilepsy proper, that the two disorders are easily separated from one another; but the author would insist upon the fact that in both cases he has given only the most typical characteristics of the diseases, and he repeats that all cases are not by any means so well defined. He would also remind the reader that the chief difficulty in making a diagnosis lies in the fact that frequently it must be made without any previous history of the case, as when a patient is brought into a hospital, in a fit, for treatment. Where the history is obtainable or where the diagnosis can be put off until the case can be studied, the question is more easily solved.

If a large number of patients suffering from these hysterical attacks be questioned between times, it will be found that the so-called *globus hystericus* becomes an almost constant precursory symptom of an attack; and if the relatives be questioned, it will often appear that they have noticed that the fall to the floor is more gentle than in true epilepsy; but this is not always so by any means. Again, the expression of the face in hysteria is, between the attacks, often very characteristic, and the surrounding atmosphere of the patient seems, even to the inexperienced, to breathe hysteria. Very commonly areas of anæsthesia and hyperæsthesia occur in these patients, and are of all degrees of intensity and limitation. Search for them generally shows their presence after attacks of convulsions, but they may exist from one attack to the other, or develop spontaneously. In nearly all cases these areas are unilateral, and may extend entirely over one-half of the body, the line of demarcation of the anæsthesia or hyperæsthesia from the sound area being clearly and abruptly defined, generally at the median line of the front and back of the trunk. (See chapter on the Skin; that part dealing with anæsthesia.) It will be called to mind that such conditions are very rare in true epilepsy. Hallucinations are far more common after the fit in hysteria than in epilepsy, and sometimes they even occur during the attacks. They are always associated with the mental state; if terror is present, rats or disgusting objects are seen, and, according to Charcot, are generally seen on the side which, during the intermissions, is anæsthetic. The pupil is more mobile in hysteria than in epilepsy, but may be contracted, normal, or widely dilated.

The following table gives, in as brief a manner as possible, the differential diagnosis between epilepsy and hystero-epilepsy, and is founded on a lecture by Professor Charcot, delivered at the Salpêtrière :

True Epilepsy.	*Hystero-epilepsy.*
Aura short.	Aura extremely prolonged.
Cry is violent.	Cry is more moderate and prolonged.
Spasms first tonic, then clonic, then followed by stertor.	Ataxic contractions, extension of limbs, turning of head, clonic movements, slight stertor.
Sometimes after fit of delirium or violent impulse or mania.	Bizarre contractions, no delirium, may be hallucinations.
Mental power is lost.	Mental power preserved.
No emotional attitudes.	Emotional attitudes.

A very useful differential point, strongly insisted upon by Charcot and Bourneville, is that in true epilepsy there is generally a very considerable rise of temperature during an attack, while in hystero-epilepsy the temperature remains normal or is only slightly raised.

In the diagnosis of true epilepsy from convulsions of a hysteroid character it is well for the physician to remember that the proportions of the two conditions in frequency of occurrence is, according to Gowers, 815 to 185 in every 1000 cases.

The differentiation of epilepsy from *uræmia* is much more readily made, for there is usually a previous history of symptoms pointing to renal trouble, as, for example, some œdema, or somnolence, or mental apathy, for some days or hours before the attack. Of course, in such cases recourse may be had to the ordinary tests for such conditions of the urine as are generally found when uræmia exists; but it is to be remembered that epilepsy and kidney disease may exist hand in hand, and that for this reason the prognosis and diagnosis are to be carefully formed and given. If in a given case a prolonged history of dyspepsia, of frequent vomiting, occasional attacks of dyspnœa, and failure of general health is found to be present, the correct diagnosis probably will be uræmia. The preservation or loss of consciousness in uræmic convulsions is variable. Generally, if the convulsion is widespread and severe, the intellection is lost; but if it be only a slight attack, consciousness may be preserved. So long ago as 1840 Bright described cases of uræmia, on the other hand, in which furious convulsions occurred without loss of consciousness, and Roberts has reported similar instances.

Fatal uræmia may also occur in a patient whose urine is apparently normal; and, in cases of chronic contracted kidney, albumin may be absent from the urine for long periods of time. The specific gravity of the urine should be carefully noted, and in very doubtful cases careful estimations of the urea be made. If the specific gravity is constantly below 1.010, the kidney will nearly always be contracted unless diabetes insipidus exists. Tests of samples of the urine taken from the total daily quantity should always be made. Another means of testing the integrity of the kidney is to administer iodide of potassium and study its elimination. It is affirmed that, after a full dose, this drug can in an hour be readily recognized in the urine by adding nitric acid and then starch; but when contracted kidney exists the iodine fails to appear or is excreted only in very small quantities. A grain of methylene-blue may also be used. (See chapter on Urine.) The temperature of the body may also be used to differentiate between uræmia and epilepsy. In 1865 Kien called attention to the fact that even when uræmic convulsions are most violent they are accompanied by a fall of temperature of as marked a character as the rise noted in epilepsy. Since then this has been confirmed by Roberts, Hirtz, Hutchinson, Charcot, Bourneville, and Teinurier.

The diagnosis between *puerperal eclampsia* and epilepsy consists chiefly in the acuteness of the attack, and the fact that with no previous convulsive history a woman becomes suddenly convulsed during the pregnant or puerperal state. This is not a place for the discussion of the identity of uræmia and puerperal eclampsia, although we believe that uræmia is generally responsible for the nervous disturbance. If the convulsions are uræmic, the temperature, according to the investigators just quoted, should fall; and according to Bourneville, puerperal convulsions are distinctly separated from those of uræmia by reason of the fact that the temperature rises with great rapidity in the very beginning of the convulsions, and there remains with great steadiness. The condition of bodily temperature can, therefore, be used to differentiate puerperal eclampsia and uræmia.

It is unnecessary to state once more that *petit mal* is but a variety or modification of *haut mal*. Nevertheless, it is useful to be able to separate it somewhat from the more severe form of the disease in the attempt to form a prognosis.

Some suppose that *petit mal* may be designated as consisting of one or two of the chief symptoms of epilepsy proper, and others have thought that the preservation of consciousness was the chief dividing line between it and fully developed epilepsy. The last idea is certainly incorrect, but it is impossible to give any outline which will absolutely separate the two conditions, so far as symptoms go. An important and useful point, first discovered by the celebrated neurologist, Weir Mitchell, is that, whereas the inhalation of amyl nitrite stops true epilepsy, the use of this drug, as a rule, increases the severity of an attack of *petit mal.*

The separation of *syncope* from epilepsy is one of the easier tasks imposed upon us. The color of the face, the weakened heart-beat, sudden loss of consciousness, and the general appearance aid us here very much.

Before closing this portion of this chapter the writer must bring forward the points to be used in differentiating epilepsy from those attacks simulated by malingerers. Often this is most difficult; and it is related by Fournier that, after his expressing an opinion that a man could always tell them apart, one of his assistants threw himself to the floor on his next visit in a pretended attack, whereupon Fournier, completely misled, exclaimed, "Poor M—; he is epileptic!" upon which the assistant, smiling, arose to his feet and refuted the statement.

Very serious injuries are sometimes submitted to by these persons to carry out their designs. The points to be looked into are: the condition of the pupils, which, in the simulated attack, always react normally; the corneal reflexes cannot be held back; the color of the face is rarely changed; and the thumbs are rarely flexed as they should be. Marc has pointed out that in malingerers the bystander can readily straighten out the thumbs and that they remain so; whereas in epilepsy they instantly become flexed again.

Suggestions as to movements are sometimes followed by malingerers, and the convulsant movements themselves generally lack the bizarre character so typical of epilepsy.

If tobacco or ammonia be held to the nose of the fraud, he generally is forced to disclose his true nature, but these drugs have no effect upon the unconscious epileptic.

The fact that in malingerers there is no rise of temperature may also serve as a differential point.

Convulsions appearing in infants or young children may result from injuries to the brain in birth, from the presence of growths, or from other distinct cerebral causes, and irritation of the alimentary canal. In these cases they may be reflexly produced.

There is one variety of infantile convulsive seizure due to meningitis, which is in itself often tubercular and associated with retraction of the head and squint; and another variety in which the symptoms very closely resemble those due to actual meningeal lesions, but in reality are quite independent of them. This condition has been called "pseudo-meningitis" or "hydrocephaloid disease," and is seen in young infants generally after attacks of severe diarrhœa. The fontanelle is depressed, the child is somnolent or comatose, and fever may or may not be present. The prognosis in the first class of cases is very bad. In the second class it is bad enough, but recovery quite often occurs if the treatment generally employed in the first class is set aside and a highly nutritious and supporting treatment is instituted.

If a child suddenly develops symptoms of acute meningitis, and has delirium, rigidity of the neck, and the major manifestations of the disease, the lungs should be carefully examined for croupous pneumonia, as this disease in children very often causes these cerebral or meningeal symptoms. Even in the adult maniacal delirium and rigidity of the neck may be present in croupous pneumonia owing to meningeal involvement.

Convulsions, which are epileptiform, sometimes occur in the later stages of Addison's disease.

Epileptiform convulsions may come on in adults as the result of *multiple sclerosis*, and they are very commonly seen in sunstroke when the patient is first attacked.

Severe convulsions have been known to follow *irrigation of the pleural cavity* after aspiration, and they may also be seen in young children suffering from whooping-cough at the time of the paroxysm.

Tetanic Convulsions.. The convulsions which are of spinal origin, namely, those that are tetanic, are the result of *tetanus* or the *ingestion of strychnine* in poisonous dose, or its fellow ignatia, and sometimes are due to hysteria. The diagnosis is aided by what has been said in the last few pages in respect to the symptoms of hysterical convulsions, and finally by the discovery of the hysterical stigmata, or the signs manifested by the skin, and, when examination can be made between attacks, of the eyes (see chapters on Skin and on Eyes).

Tetanus convulsions and strychnine-poisoning are to be separated from one another by the fact that in tetanus the locking of the jaws comes first, while in strychnine poisoning it comes last. The convulsions of tetanus rarely, if ever, completely relax, while those of strychnine do have periods of complete relaxation. There is a different history in each case: in one, perhaps, of an injury, as of a nail run into the foot; in the other, of a dose of poison having been swallowed.

The differential diagnosis between strychnine-poisoning and hysterical convulsions is more difficult. The convulsions are rarely so persistently tonic in hysteria as in strychnine-poisoning, and the peculiar expression of the hysterical face is often seen in this disease. The history of the patient, if obtainable, will throw much light on the case and aid very materially in the separation of the two conditions.

Tetany. When a patient is seized with sudden and symmetrical tonic spasms of the hands, extending to the upper arms and shoulders, so that the fingers are flexed at the metacarpo-phalangeal joints and extended in the phalangeal joints, and the lower limb is flexed, while the legs are extended and the toes are flexed, the condition is one of *tetany*. (See chapter on the Hands and Arms, "accoucheur's hand.") It is most commonly seen in hysterical cases and has no relation to true tetanus. Pressure on a nerve-trunk or bloodvessel will often produce an attack in such persons, and this is sometimes called "Trousseau's symptom." The pressure must be applied for several minutes in some cases, and the best place to apply it is the bicipital sulcus or the crural sulcus. Sometimes pressure on the brachial plexus or on the popliteal space will be provocative of an attack. It is not a constant symptom, but pathognomonic if found. Another equally useful diagnostic sign is called Chvostek's facial symptom. This results from the fact that the facial muscles are irritable, so that when they are tapped by the finger-tip, or a rubber hammer, contraction results. The tapping is usually applied over the zygomatic arch in its anterior portion, and this will result in a spasm of the upper lid of the eye and the alæ nasi. In other cases stroking the area over the parotid will have the same effect. The muscles in tetany also have an increased electrical excitability. (Erb's symptom.)

It is worthy of note that both Trousseau's and Chvostek's symptoms are sometimes met with in rhachitic children, particularly if they have craniotabes. (See chapter on the Head.) Laryngismus stridulus will often be found associated with tetany and rickets.

Convulsions limited to a few muscles or more widespread in character may appear as symptoms in *acute yellow atrophy of the liver;* but the peculiar symptoms of this disease render easy the diagnosis of their cause.

Convulsions may also arise from *hæmatoma* of the dura mater (internal hemorrhagic pachymeningitis), but the diagnosis from those due to cerebral hemorrhage is practically impossible.

Spasms. General spasms, in distinction from convulsions, are represented by chorea in its various forms, and by saltatoric and palmic spasm, paramyoclonus multiplex, and the occupation-neuroses. There are other localized spasms from nervous disease, such as facial spasm and wryneck, athetosis, and post-hemiplegic chorea. Some of these conditions will be found discussed in the chapter on the Hands and Arms and that on the Face and Head.

When a patient is afflicted more or less constantly and more or less universally by disordered, irregular, jerking movements which throw the part or parts affected into unusual positions, which are not, however, maintained even for a moment, the condition is probably *chorea minor*. Often the speech is seriously disturbed by reason of the choreic movements of the lips and tongue or jaws, and some loss of power may be manifest in certain muscles. This true chorea or St. Vitus's dance may affect the whole body or only one arm or leg, but generally it is diffused. Commonly it ceases at night when the child sleeps, but it often persists day and night, and then becomes a serious malady, because of the exhaustion produced. It often follows fright, prolonged bad weather, and other causes which may upset the nervous balance of the child. Chorea in childhood is so characteristic in its manifestations that it can be readily recognized in most cases; but it sometimes has to be separated in adults from disseminated sclerosis, progressive muscular atrophy, hysteria, and Friedreich's ataxia. The movements in disseminated sclerosis are, however, fine muscular tremors, instead of minor jerking movements; and there are present nystagmus and scanning speech in sclerosis, but not in chorea. Again, in progressive muscular atrophy there is fibrillary muscular tremor, but not twitching of a marked form, and the muscles are wasted. In hysteria the muscular movements are rarely choreic, and the presence of changes in the color-fields and the other stigmata of hysteria (see chapters on Skin and on Eyes) renders a diagnosis of the latter condition easy. Friedreich's ataxia is to be separated from chorea by the scanning speech, scoliosis, slow incoördinate movements, and the family history of the disease.

Rarely when there is some paresis with chorea, the patient may present symptoms of acute poliomyelitis; but the paralysis in the latter affection is more marked, and there are no movements in the affected muscles, such as occur in chorea.

Chorea insaniens is a violent form of ordinary chorea associated with mania, which is not to be confused with choreic movements occurring in the choreic insane.

Choreic movements sometimes come on in the aged, and must be separated from paralysis agitans and senile trembling. This is possible by the fact that in paralysis agitans the movements are tremors, and there is loss of power with the peculiar facial expression ("Parkinsonian visage") and a hurrying gait (festination). Senile trembling is usually an affection limited to the head, and consists in a tremor and not in marked twitching. (See chapter on Hands and Arms, part on Tremors.)

A rare form of chorea has been called *Huntingdon's chorea.* It occurs in adults about the age of thirty to forty years. It is hereditary; that is, there is generally a history of the same trouble in the ancestors of the patient, and finally as it progresses psychical disturbances ensue. Irregular movements first appear in the hands, which movements become markedly incoördinated, the arms are thrown about in excessive and rapid jerkings, and when the infection involves the legs a characteristic gait is developed of a dancing or "hop, skip, and jump" character. Sometimes, early in the malady, the movements can be controlled by the will. The face passes through slowly formed grimaces, and the gait may be staggering. The speech becomes indistinct, and enunciation is not clear. Finally, dementia closes the scene. The movements of Huntingdon's chorea are not sudden as in true chorea; it is a disease of adult life, and mental disturbance is a prominent symptom. These facts separate it from ordinary chorea.

When the patient involuntarily bends over in a profound bow the cause of his movements may be rhythmical contraction of his abdominal muscles, producing the so-called *salaam convulsions* or chorea major.

A still more rare malady is *electric chorea* or "Dubini's disease," in which the muscles of the arm and then the leg on the same side are affected with a sudden muscular spasm or shock, such as is produced by the electrical current. · Wasting of the affected muscles, loss of faradic irritability, occasional epileptic convulsions, and rarely elevation of temperature come on. The disease is a fatal one, and generally occurs in malarial regions in Italy. Under the same name of electric chorea Bergeron has described a state of rhythmical muscular spasm which usually ends in recovery.

When a condition of clonic muscular spasm affecting the trunk, limbs, and perhaps the neck, is present, the hands and toes being uninvolved, as a rule, the possibility of the presence of *paramyoclonus multiplex* is to be considered. The spasms in this rare disease are bilateral and occur at intervals, often only on an attempted movement or speech. So violent are the muscular contractions in some cases that the patient may be thrown to the ground or, if in bed, to the floor. These movements may vary from three or four to 120 per minute, but are generally about 50 per minute. The symmetrical bilateral involvement, the fact that the movements are not choreic in character, and that the patient is a male, are to be remembered in making the diagnosis. The ultimate prognosis is favorable unless the movements are so constant as to cause exhaustion. Care must be taken not to confuse hysterical movements with this condition. The bilateral movements which affect only the larger muscles, and the fact that paramyoclonus multiplex is nearly always seen in the male, separate it in part from hysteria, while the hysterical stigmata when they are present will point to hysteria as the cause of the disorder.

Sometimes a patient will be met with in whom, when he attempts to stand, the leg muscles first become rigid and then are thrown into violent contractions, which cause him to jump up and down, or he may be thrown to the floor. This condition is called *saltatoric spasm* or "jumpers." It is to be separated from the condition of the legs seen in lateral sclerosis of the cord by the fact that in the latter disease the legs become spastically stiff on attempting to use them, from Huntingdon's chorea in that voluntary movements with the hands may be performed perfectly, and from chorea minor by the absence of small incoördinated twitchings.

Such a patient will often act on suggestions or in imitation of the acts of other persons or of animals.

Some writers confine the term "saltatoric spasm" to those cases which possess no imitative features. In such cases the disease is far more moderate in its manifestations.

Quite distinct from these clonic spasms of the muscles brought on by attempted movement is that in which the muscles become tonic on attempted movements. At first they are stiff and slow in their movements, but ultimately develop a tonic spasm, so that walking is at first almost impossible, but the limbs limber up on exercise. This is a rare affection, called *Thomsen's disease,* or one of the forms of *myotonia congenita.* (See chapter on Feet and Legs.)

Forced gyratory movements of the body are sometimes seen as the result of a lesion of the middle peduncle of the cerebellum.

CHAPTER V.

HICCOUGH, VOMITING, REGURGITATION, AND THE CHARACTER OF THE VOMIT.

Due to uræmia—Cerebral lesions—Intestinal obstruction—Peritonitis—Cholera—Gastric disease—Hepatic disease—Poisons—The appearance of vomit.

HICCOUGH or singultus may or may not possess considerable clinical significance. Often it arises from slight indigestion. In other cases it is produced by the drinking of sparkling wines or waters. When hiccough becomes persistent it is a symptom to be regarded with interest, for if it continues for a long period of time it is usually significant of hysteria or uræmia, while if it develops in a patient exhausted by some prolonged or severe illness it shows deep depression of nervous tone, and is itself dangerous because of the exhaustion it speedily produces. Sometimes it is said to be an annoying symptom after passing catheters or bougies in cases of stricture in the urethra. Hiccough develops in peritonitis, and is a most distressing symptom. It is also seen in cases of intestinal obstruction or when abdominal growths are developing. Singultus also takes place in some cases of cerebral hemorrhage, in myelitis affecting the upper parts of the spinal cord, and in very rare instances because of severe mediastino-pericarditis involving the phrenic nerve. It also occurs as a result of central nervous irritation in persons suffering from advanced anæmia, and in cases of suppurative hepatitis.

VOMITING is the act by which the contents of the stomach are forcibly expelled from this viscus through the cardiac orifice, the œsophagus, the pharynx, and the mouth. The vomiting-centre in the medulla oblongata gives rise to the necessary nervous impulses, and is provoked to this by direct stimulation or by reflex irritation. Thus in uræmia the vomiting sometimes encountered is the result of irritation ol the centre by some unknown poison. When apomorphine is given the centre is also stimulated. Centric vomiting is also caused by the administration of anæsthetics, notably ether and chloroform. On the other hand, gastric, intestinal, or other abdominal disorders may reflexly produce very persistent emesis, and for these reasons vomiting is of considerable diagnostic importance.

As vomiting is produced by many maladies, it is a symptom frequently met with. It occurs with a certain degree of constancy as a complication or symptom of uræmia, diabetes, apoplexy, brain-tumor, brain-abscess, Ménière's disease, tubercular meningitis, hysteria, intestinal obstruction from all its various causes, gastric and intestinal indigestion, gastritis, gastric ulcer, gastric cancer, peritonitis, nephritic colic, hepatic jaundice, hepatic colic, in cholera, yellow fever, and a host of other ailments. Sometimes the onset of one of the acute infectious diseases of childhood is characterized by vomiting. Not infrequently this symptom associated with diarrhœa masks the presence of the real cause of the illness, as in some cases of croupous pneumonia.

The vomiting of *uræmia* may be one of the earliest manifestations of renal disease, and its presence, when persistent in the absence of local gastric or other causes, should always lead to an examination of the urine, since valuable time may be lost if the patient is considered to be suffering from some slight indiscretion in diet. Its association either as a preceding, concomitant, or consequent symptom of convulsions renders a diagnosis of uræmia probable, while a history of uræmic amaurosis, colliquative diarrhœa, and failure of the general health will be very important points in reaching a decision. No pathognomonic symptom of uræmic vomiting exists unless we consider the urinary evidence a symptom, but in some cases the vomited matters smell strongly of carbonate of ammonium, resulting from the decomposition of the urea which has been eliminated from the blood into the stomach by the gastric mucous membrane. Uræmic vomiting is, therefore, not only due to centric irritation by a poison in the blood, but to irritation of the stomach by the urea which is excreted into it. Diabetes comparatively rarely produces vomiting by the toxæmia which it causes, but in any case the urinary examination and polyuria decide the diagnosis.

When vomiting results from *cerebral hemorrhage*, *embolism*, or *thrombosis*, the focal or hemiplegic symptoms characteristic of apoplexy are present. Possibly the vomiting is more indicative of hemorrhage than of plugging of the vessel. A sudden attack of vomiting in a previously healthy man of advanced years, or in one who is young but has a specific history, should raise the question as to the possible presence of one of these lesions; provided, of course, that ordinary gastric disorder is not present as a cause.

The vomiting due to *cerebral tumor* is generally preceded by the characteristic severe and constant headache, vertigo, a slow pulse, impaired memory, and sometimes by epileptiform convulsions. Further than this, the important diagnostic ocular symptom called "choked disk" of the optic nerve is to be sought for, and if found is of great positive value. Tumor of the brain, if near the base, often causes, too, involvement of the various cranial nerves. (See chapter on the Eye.) The vomiting of cerebral tumor is independent of taking food, and commonly comes on early in the morning, thereby differing from some of the forms of vomiting due to gastric disorder. The vomiting arising from *cerebral abscess* has symptoms precisely like those just named, so that a differential diagnosis is almost impossible. The history of injury or of an infectious process producing a secondary brain-abscess may point to this cause of the vomiting: the real points of difference are that in abscess choked disk is rarely seen, fever is commonly present, and the cranial nerves generally escape. When *purulent meningitis* produces vomiting it may be impossible to tell whether this symptom is due to it or to an abscess, as the purulent collection may be localized. Vomiting sometimes results from *profound cerebral anœmia* of an acute type due to hemorrhage, in fainting or in chronic anæmia, as in chlorosis. Generally, however, the symptom is only a constant nausea. The presence of great pallor and other evidences of anæmia aid in the diagnosis, but it must not be forgotten that some severe anæmias are accompanied by febrile movement and by marked choked disk, which should not mislead the physician into a diagnosis of cerebral tumor.

When vomiting is due to *cerebellar tumor*, the diagnosis is aided by the presence of vertigo, the peculiar staggering gait, and finally by evidences of choked disk, on ophthalmoscopic examination, with disordered vision.

The vomiting of *meningitis* is quite frequently an early symptom, but it also often occurs later in the disease, and is caused by the meningeal irritation, and not by any condition of the stomach, unless that viscus has been disordered by the unwise use of drugs. This form of vomiting can nearly always be separated from that due to other causes by the excessively severe headache, chiefly of an occipital type; by the pain in the nape of the neck and in the spine; by the rigidity of the dorsal muscles, so that opisthotonus may be caused in severe cases; and, finally, by the disordered functions of the cranial nerves, as a result of which there are found trouble in the oculomotor nerve, strabismus, double or single ptosis, slowly reacting pupils, which may be unequal, nystagmus, and sometimes facial contractions due to involvement of the facial nerve.

Vomiting due to *acute miliary tuberculosis* often comes on at the very onset of the malady, and is associated with obstinate constipation, or, on the other hand, active diarrhœa; but the fever, the very rapid pulse, the wasting of the patient, the possibly present physical signs of tuberculosis of the lungs, and, very important, the peculiarly severe dyspnœa, for which no adequate cause can be discovered on physical examination, all point to the general infection. If a skilful examination of the eye can be made with the ophthalmoscope, the choroid may be found to be studded with tubercles.

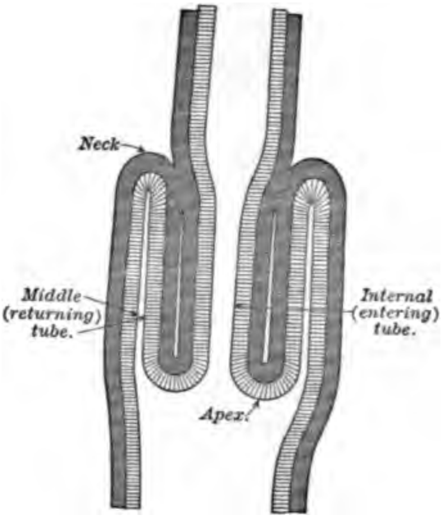
The reflex forms of vomiting are very numerous, and depend chiefly upon organic and functional disorders of the abdominal viscera. In some of these conditions vomiting is of little importance, except for its gravity if the patient is exhausted; in other words, it is simply a disagreeable symptom. In others the symptom vomiting is of considerable diagnostic value as indicating the grave mischief which produces it. One of the most important of the latter conditions is *intestinal obstruction*, whether it arises from intussusception, constrictions by bands, volvulus, or impactions.

In *intussusception* vomiting is practically a constant symptom, occurring with the sudden pain, or, at times, even preceding it. In children it continues till shortly before death, and is rarely feculent.

In the adult, and in the chronic form, there may be complete absence of vomiting, though this is certainly exceedingly rare. Leichtenstern takes exception to the statement that the seat of obstruction is indicated by the period at which vomiting is developed. The ileum-invagination is most frequently accompanied by early vomiting, not because of its seat, which is usually but little removed from the ileo-cæcal valve, but because it is commonly obstructive. The vomiting, both in time of development and in nature, will depend, not upon the seat of the trouble, but upon the presence or completeness of obstruction, and may be early if the obstruction is absolute in the sigmoid flexure, and feculent if the occlusion is in the upper part of the ileum.

The pain is usually sudden, violent, diffuse, or, if localized, usually centred in the ileo-cæcal or umbilical region. After a few hours in children, a much longer interval in the adult, the pain ceases, often as suddenly as it commenced, and there is an interval in which there is little to suggest that the pathological condition still continues. This is followed by a return of the pain, the paroxysms becoming more violent and prolonged, the intervals less marked as the disease progresses, or in the adult, if it passes into the chronic form, and intervals even of days may elapse between the paroxysms. The pain is frequently accompanied by tenderness, but this is an exceedingly variable symptom, and at times pressure seems to relieve the pain.

FIG. 231.



Intussusception.

Blood-stained mucous evacuations are a symptom of intestinal obstruction which, in children, is rarely wanting. Of 108 cases of invagination in the first year of life this symptom was absent in but four. It occurs within a few hours of the first attack. At the first the discharge is of blood-stained feces; later, if obstruction is developed, of blood and mucus, and is usually exceedingly offensive. In children diarrhœa is common throughout the whole course of the case. At times, following complete constipation and feculent vomiting, there will suddenly appear copious evacuations from the bowel, mingled with blood, in which may be found evidences of the necrosed intussusceptum. Where this slough is extensive it may be lodged in a lower portion of the bowel and cause fatal obstruction.[1]

In connection with the muco-sanguinolent evacuations the tenesmus or straining is a symptom so common that it is of some diagnostic import. That it is not dependent upon the character of the evacuation is shown by the fact that it is present in cases of complete obstruction. Brinton has shown that this symptom is seldom developed except in the ileo-cæcal and colon invaginations.

A much rarer condition, and one which Leichtenstern ascribes to the secondary effect of intense tenesmus, is a patulous condition of the anus due to paralysis and dependent upon invagination of the descending colon and rectum. This is never produced by invagination of the ileum.

Leichtenstern's statistics show that a tumor can be felt either through the parietes or by rectal examination in 52 per cent. of all cases. In the first year of life this most important diagnostic sign was present in 63 per cent. The tumor is usually felt in the left iliac region, or by the finger passed into the anus. The ileo-cæcal invagination is most frequently accompanied by demonstrable tumor; the ileum-invagination exhibits this symptom with less frequency.

Many authors have noted that the tumor varies in size and consistency from time to time, now, during an acute paroxysm of pain, being hard, knotty, and plainly perceptible, shortly afterward eluding the most careful search. Duchaussoy has described two distinct movements which can often be perceived in the tumor, namely, the erectile and the vermicular motion.

Distention of the abdomen is not of great significance, because it is often absent. In children especially it may not appear at all, or may appear only just before death. In adults, in whom obstruction is more common, it may become as well marked as in obstruction from any other cause.

Dance calls attention to an inequality in the shape of the abdomen dependent upon the meteorism, and in view of which he states that the seat of obstruction can often be inferred. But few authors, however, have been able to profit by his observation.

In the chronic form of invagination the symptoms are less violent in onset; there may be nothing more characteristic of the attack than recurring paroxysms of pain, meteorism, and obstruction, with symptoms of intestinal stricture constantly manifesting themselves. These cases terminate either in cure by reduction or by extrusion of a slough, or perish from exhaustion, inanition, or in the course of an acute attack. In over one-half of the recorded cases a correct diagnosis was not made.

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[1] For much information on the subject of intestinal obstruction see the Fiske Fund Prize Essay of the Rhode Island Medical Society for 1890, by Dr. Edward Martin and the author.

The additional symptoms upon which a diagnosis of vomiting from intussusception is to be based are the acute onset of colicky pain, and its intermittent character; passages from the bowels containing blood and mucus; the presence of tumor, commonly in the left iliac region, or felt through the anus, varying in size and consistency from time to time, with an erectile or vermiform motion; and the ordinary obstruction-symptoms. The diagnosis is further confirmed if there are violent peristalsis and tenesmus, and if these symptoms occur in an infant.

According to Leichtenstern, Bryant, and others, 40 per cent. of all cases of intestinal obstruction are due to intussusception, and this condition is most common in the first year of life, after which it becomes more and more rare until the fortieth or fiftieth year, when it increases in frequency. The prognosis is bad, the mortality varying from 73 to 90 per cent. unless early surgical relief is given.

Internal strangulation by bands occurs in from 25 to 30 per cent. of the cases of obstruction of the intestine, and affects males most commonly between twenty and forty years of age. In 133 out of 151 cases the small intestine was involved. The typical symptoms are as follows:

1. Sudden, agonizing pain, constant, and located about the umbilicus, with paroxysmal increments.

2. A rapid, weak pulse and subnormal temperature. This is nearly constant in the early stages of the attack; later on, when local or general peritonitis develops, the temperature and pulse may assume the features characteristic of inflammation.

3. Vomiting. First of the contents of the stomach, then of bile, and, finally, in a large percentage of cases, of feculent matter. The feculent vomiting rarely appears before the third day, and in cases running a very acute course death may ensue before this symptom has time to develop. The vomiting is constant and gives no relief to the patient.

4. Constipation. Exceptionally there may be one or two passsages representing the contents of the bowel below the seat of obstruction;

after that the constipation is absolute, not even flatus passing by the anus. Treves has suggested that the evacuations sometimes observed toward the termination of the case, and not due to the relief of obstruction, may be dependent upon the beginning of peritonitis.

5. Tympanitic distention. Where there is a large segment of gut involved in the strangulation its early distention may give rise to a localized abdominal enlargement, which is exceedingly suggestive as to the cause of the attack. In general, the meteorism is not marked except when peritonitis supervenes.

Since in the large majority of cases the obstruction is localized in the lower portion of the small intestine, the primary distention will be observed in the middle abdominal region—*i. e.*, the epigastric, umbilical, and hypogastric areas. Laugier claims by this symptom to locate the obstruction with some certainty.

The violent peristalsis and repeated vomiting prevent the extreme meteorism characteristic of intestinal paralysis.

6. Localized tenderness and percussion-dulness. When present these signs are of exceeding great importance, since they denote the position of the strangulated bowel.

Exceptionally a tumor may be felt, formed by the congested gut or the matting together of the intestinal coils.

The urine is diminished in quantity and may be suppressed. In acute strangulation it commonly contains albumin, and it is stated that this is of diagnostic value.

In this connection the history is of great importance.

Other congenital deformities would suggest the possibility of Meckel's diverticulum as a cause.

A preceding typhlitis, pelvic peritonitis, or severe abdominal traumatism would respectively assign an adherent vermiform appendix, peritoneal bands, or rents in the omentum or mesentery as the causative agents in the production of the symptoms.

The age of the patient should also be considered, since this form of obstruction usually occurs between the twentieth and fortieth years.

The sudden onset of violent, persistent pain, subnormal temperature, and frequent pulse, the obstinate, absolute constipation, the persistent, repeated vomiting, becoming fecal, and the rapid course of the disease, all point to internal strangulation.

Auscultation of the abdomen is at times of value, a sound compared to the click of the water-hammer being heard most distinctly at the point of obstruction.

Palpation and percussion should not be omitted, as thereby the seat of obstruction has been distinctly located.

Volvulus is the most frequent form of intestinal obstruction after intussusception and that due to strangulation. Vomiting occurs, but is not so constant a symptom as in those forms first named. Thus it occurred in from 8 per cent. in Brinton's statistics to 2.5 per cent. in those of Treves, and 4 per cent. in Martin's and the author's. It is nearly always seen in men in middle life. The vomiting is rarely fecal, is very slight in many cases, and sometimes does not appear at all.

Vomiting, on the other hand, is quite commonly seen in the cases of *obstruction from impaction* or obstruction from foreign bodies. This underlying cause of the emesis can be diagnosed by the history of a foreign body having been swallowed, of attacks of hepatic colic, or, where a gallstone ulcerates through into the bowel, or of some local peritonitis about the region of the liver. A history can commonly be elicited of sharp, colicky pain, of partial obstruction, and of vomiting. The distention is slight, the amount of systemic shock far less than in other forms of obstruction, and the duration of the attack somewhat longer than usually obtains in this class of affections. The symptoms of obstruction are frequently only partial, the vomiting being moderate in amount and not stercoraceous, the constipation not being absolute.

Except in the case of enteroliths and very large foreign bodies a tumor can rarely be felt.

It is often impossible to diagnose this form of obstruction from that depending upon a narrowing of the lumen of the bowel, such as is produced by cancer or stricture. The previous history is always of great importance. The presence of indican[1] rather than albumin in the urine, the comparative mildness of the attack, the moderate meteorism, and the slow course of the disease, all help to exclude internal strangulation or volvulus. It is, however, mainly upon the history that the diagnosis will be founded.

In this connection it is to be remembered that *hemorrhagic infarction* of the intestine may produce symptoms similar to those just described, namely, pain in the region of the navel or more generally throughout the belly, fecal vomiting, diarrhœa, and bloody stools. Actual obstruction may be present. Search should be made for a source from which an embolus may arise, as, for example, valvular cardiac disease, and if this is found the likelihood of infarction being present is increased.

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[1] For the test of indican, see chapter on the Urine.

Vomiting, loss of appetite, thirst, cough, hectic fever, and sweats, with the development of marked cachexia, sometimes occur from *obstruction of the rectum.*

When vomiting arises from *general peritonitis* it is often one of the earliest symptoms of the malady. It is almost always present, and is often a very severe symptom, and is associated with or replaced by a constant retching, which adds to the exhaustion of the patient. At first it may only follow the swallowing of food, but often it occurs without such a cause, and after the stomach is emptied of its ordinary contents glairy, watery mucus is expelled, which is often of a distinct greenish tint. The great tenderness of the belly in acute peritonitis, the moderate fever, the rapid pulse, the anxious face, and the cold skin as collapse approaches, all render the diagnosis easy; but it is to be remembered that the distention of the belly by an overfilled bladder or pregnant uterus may mislead the physician into thinking that peritonitis is present because of the swelling, the pain, and the vomiting. Vomiting is not a severe symptom of appendicitis unless the peritoneum has become involved in the inflammatory process, although it may occur once or twice when the pain in the appendix is most severe. The localization of the symptoms in the neighborhood of the appendix makes the diagnosis possible. (See chapter on Abdomen.)

When vomiting occurs in typhoid fever, it is usually a symptom of bad feeding or imperfect digestion, and is rarely of grave importance except under two conditions. The first of these is when it occurs as a result and symptom of intestinal perforation, an accident commonly seen late in the disease; and, second, when it takes place as an obstinate and exhausting symptom after the fever has practically passed by, from unknown causes, probably reflex in character. The symptoms of perforation other than vomiting can be found in the chapter on the Abdomen and Abdominal Viscera.

Vomiting as a symptom of *cholera* is accompanied by serous diarrhœa of profuse character, by the development of collapse, cramps in the muscles, anuria, and great circulatory failure. It should be separated from the vomiting due to cholera morbus or severe indigestion, antimonial poisoning, and arsenical poisoning. Cholera morbus is to be separated from cholera, first, by the absence of the comma-bacillus in the stools; second, by the fact that there is a history of exposure to cold or damp, or bad food; third, by the absence of an epidemic; and, fourth, by the fact that its manifestations are milder.

No one can be skilful enough to separate symptoms of poisoning by antimony from those due to cholera, for they are identical in every way. Nothing but the history of the ingestion of the poison and the discovery of antimony in the secretions can prove the case to be one of antimonial poisoning, particularly if an epidemic of cholera is present.

In *arsenical poisoning* the association of vomiting with bloody stools separates the symptoms from those of cholera.

Vomiting is often a very severe and early symptom of *cholera infantum* (see chapters on Abdomen and on Bowels and Feces), and it occurs in attacks of *true dysentery* as a common symptom, when its underlying cause is readily discovered. (See Abdomen.)

The diseases of the stomach causing vomiting are cancer, ulcer, gastritis, catarrh (acute and chronic), true gastritis, and dilatation.

The vomiting of *gastric cancer* at first consists in the expulsion from the stomach of its contents—mixed particles of food, mucus, water, and sometimes bile. The vomit may be tasteless or sour from fermentation, and may have an offensive odor from similar causes. Often it contains blood, either in bright-red streaks or as a brownish-red fluid, or in similarly colored clots, which may be brown when they have been in the stomach for some time. Often the exuded blood, changed by mixture with the stomach-contents, looks like coffee-grounds, producing "coffee-ground vomit." This coffee-ground vomit is not pathognomonic of gastric cancer, but is very characteristic of this disease. Microscopically the vomited materials are seen to consist of particles of food, yeast-cells, cocci, and broken-down blood-corpuscles. (For the other symptoms of gastric cancer, see chapter on the Abdomen.)

Coffee-ground vomit is also sometimes seen in cases of *locomotor ataxia* following a gastric crisis. Roux asserts that overdoses of iodide of potassium may produce a gastric crisis in locomotor ataxia by irritating the stomach.

Vomiting due to *gastric ulcer* is preceded by pain, and is generally brought on by taking food, and so occurs soon after eating. The food is, therefore, only slightly digested, and evidences of fermentation are absent to a great extent. If blood is present, it is nearly always bright red and in considerable quantity, and indicates that a hemorrhage has recently taken place from the surface of an ulcer. Very large hemorrhages into the stomach may cause vomiting by irritating and distending this viscus. The history of vomiting after eating, the presence of blood in the vomit, the pain in the stomach, the age of the patient (generally twenty to thirty years), the sex (generally female), and the hyperchloric acidity, combined with the other symptoms (see chapter on Abdomen), complete the diagnostic array of facts.

There are, however, other causes of vomiting of blood or hæmatemesis than gastric ulcer and cancer. Thus it occurs from obstruction to the portal circulation from *hepatic cirrhosis*, and from growths and splenic affections which result in varicosity of the bloodvessels of the stomach. Hæmatemesis also follows severe blows, kicks, and other injuries to the epigastrium. Sometimes it takes place in cases of heart disease in which there has resulted hepatic engorgement with secondary gastric congestion, and it may be developed in small degree by any form of violent vomiting which strains the stomach, particularly if an irritant substance has already destroyed the mucous membrane. Again, hæmatemesis is seen in scurvy, typhus, yellow fever, and acute yellow atrophy of the liver, as a result of breaking down or destruction of the coats of the vessels. Sometimes it is seen in cases of dengue, in influenza of the epidemic type, and in relapsing fever. Hæmatemesis may also occur in purpura hæmorrhagica, in hæmophilia, and as a result of vicarious menstruation. In malarial fever of a severe character the dark-colored vomit is generally due to bile, but it may be due to exuded blood. Such a case is reported by Boon as occurring in a child.

Care should always be taken that the physician is not misled by the vomiting of swallowed blood into a diagnosis of gastric hemorrhage from any of the causes just named. It may enter into the stomach from the pharynx, as after epistaxis, or blood may be swallowed by a malingerer. Hæmatemesis is to be separated from hæmoptysis by the fact that in the latter there are physical signs in the lungs, the sputum is frothy and bloody, there is absence of retching or vomiting-movements, and the blood is bright red in hæmoptysis oftener than in hæmatemesis.

Under the name of *melæna neonatorum* there is a condition of hæmatemesis occurring in children within the first few days or weeks of life. This condition has been thought by Leube to be due to gastric and duodenal ulcers, and his views are indorsed by Buhl and Huhn, Spiegelberg, Binz, and Landau. In one of the latter's cases thrombosis of the umbilical vein was present, and it has been thought that when expansion of the chest takes place in the newborn child small clots may escape from this vessel through the ductus arteriosus into the descending aorta and gastric arteries, and thus cause an ulcer of the stomach by embolism.

The vomiting of *acute gastric catarrh* is generally seen in children, and is often preceded by great nausea. The contents of the stomach are first gotten rid of, then mucus, water, and bile may be ejected, and finally exhausting retching ensues if the attack is severe. The tongue in such cases is coated and dotted with red spots from the enlarged fungiform papillæ, and the epigastrium is tender on pressure. There may or may not be fever and looseness of the bowels. The attack usually follows indiscretions in diet or exposure to cold.

Vomiting from *chronic gastric catarrh* is usually a condition met with in adults, and when seen in the male is most frequently the result of a frequent use of alcoholic beverages to excess. In women it often develops from excessive tea-drinking associated with errors in diet. When due to alcoholism, the vomiting is often present only in the morning before or after taking food, and then is called the "morning vomiting of drunkards." (See chapter on the Tongue.)

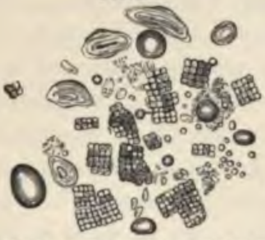
Vomiting due to *true gastritis* or inflammation of the stomach in its deeper layers is very rare, except as a result of the ingestion of an irritant poison or hot liquid.

Perhaps the vomiting occurring in *dilatation of the stomach* is more typical in its character than any other. This act is always a prominent symptom of gastric ectasy, the matters vomited being often greenish and extremely fetid, and nearly always profuse in amount. Examination of the ejecta will generally show food swallowed days before, owing to the imperfect digestive action of the stomach, and this very inability of the stomach to act on the food generally gives, for a long period of time, a sense of weight and fulness often amounting to pain, and complained of bitterly. There is tenderness over the epigastrium on pressure, and the displacement produced by the palpation often brings on either acid or yeasty eructations or even the vomiting already named. Nausea preceding the vomiting is by no means common, there being simply a gush of foul liquids from the mouth. After such an occurrence the vomiting fails to recur for from twenty-four to forty-eight hours, or perhaps for a week—*i. e.*, until the viscus becomes overladen once more. The gases which are given off on eructation are exceedingly acrid, nauseous, and bitter. Sometimes they are offensive, but more rarely odorless. They are often inflammable, and consist of oxygen, nitrogen, hydrogen, and carbonic acid. Sometimes sulphuretted hydrogen is present in large quantity. The reaction of the vomit is almost always acid, lactic and butyric being the acids most commonly found, but the normal hydrochloric acid is usually absent. Fibres of meat or masses of semi-digested and semi-decomposed food can be seen by the naked eye or under the microscope, and sarcinæ and many forms of bacteria swarm in the mass. Particular search should be made for the yeast fungus, *torula cerevisiæ*, the presence of which is a certain evidence of active fermentation.

(For further information in regard to the symptoms of gastric dilatation, see the chapter on the Abdomen.)

Vomiting due to gastric dilatation should not at once lead the physician into a diagnosis of stenosis of pylorus from growth or constrictions in this part of the stomach, or from innate feebleness of the gastric walls, for similar conditions may follow growths of the pancreas, which by pressure occlude the pyloric opening (see chapter on Abdomen). If cancer is present in the stomach, an examination of the gastric contents after a test-meal will fail to reveal the presence of hydrochloric acid.

FIG. 232.



Sarcinæ ventriculi, with starch-granules and oil-globules, from vomited matters. (OTTO FUNKE.)

Sarcinæ ventriculi are found not only in the frothy, dirty-looking vomit of gastric dilatation, but in that of chronic gastric catarrh, cancer, and ulcer of the stomach. If iodine or iodide of potassium is added to the vomit, the sarcinæ become mahogany-red or brown, and are easily recognized, occurring in squares which are separated by dividing lines. (Fig. 232.)

Vomiting also arises from *neuroses of the stomach* in several forms. Thus it is frequently seen in hysteria, in neurasthenia, pregnancy, and sometimes occurs in the form of what Gee has called "cyclical vomiting." It also comes on in association with gastric crises in locomotor ataxia. The vomiting of *hysteria* is generally characterized by its persistent character, often lasting for months, and yet the patient often maintains her normal weight to a surprising degree, either because the food which is taken is only vomited in small part or because she surreptitiously obtains food when her attendants do not know it, which she retains. It is generally associated with so many of the hysterical stigmata as to be readily diagnosed. The vomiting of *neurasthenia* is seen in both sexes, and is particularly apt to follow any nervous muscular exertion. Thus in one case of the writer's even short railroad journeys taken by an overworked man produced attacks of spinal tenderness with vomiting, which lasted several days. In some neurotic cases the vomiting takes place as soon as the food is swallowed. The vomiting of *pregnancy* is usually a morning vomiting, though it may persist all through the day. It has no particular diagnostic features in itself, save that there are rarely any signs of gastric indigestion. The presence of pregnancy, of course, makes the diagnosis clear; but in such cases, if the pregnancy is advanced, the physician should always examine the urine, since the ordinary vomiting of pregnancy is a symptom of the first few months, while that occurring later on may indicate grave renal complications. (See Uræmia, in chapter on Coma and in chapter on Convulsions.)

The *cyclical vomiting* already mentioned is generally seen in children, and is of rare occurrence. It is characterized by attacks of vomiting recurring after intervals of uncertain length, during which the patient may seem entirely well. The attack may last from a few hours to a few days. There are often pain in the epigastrium and constipation. Sometimes retching is the main symptom. It is generally seen in neurotic patients.

In order to determine that the discolored vomit of any case is due to blood, a microscopical examination for the corpuscles must be made, and if these are greatly altered a chemical test may be used. (See further in this chapter.)

The development of vomiting with sudden pain in the abdomen, resembling colic, which fails to yield to ordinary remedies, and is associated with sources for an embolism, should lead the physician to a consideration of possible *embolism in the superior mesenteric artery*. It may also be a symptom of Raynaud's disease.

This condition should not be confounded with the vomiting of *acute pancreatitis*, in which colicky pain in the epigastrium, deeply seated and extending to the right shoulder and back (see Hepatitis, in this chapter), and great restlessness, præcordial distress, dyspnœa, and faintness are present. The matters vomited are greenish, clear, and viscid, and the efforts at vomiting increase the pain. There is no jaundice, and death soon comes to the relief of the patient.

As an early diagnosis of acute pancreatitis may permit surgical interference, with possible recovery of the patient, the diagnosis is important. The mistake commonly made is to consider the case one of intestinal obstruction.

Vomiting of a peculiar character is always present in *phosphorus poisoning*. The symptoms associated with ingestion of the poison may not come on for some hours. At the end of that time the peculiar taste of phosphorus may be noticed in the mouth, the breath is heavily laden with its odor, and burning pain in the œsophagus, stomach, and abdomen ensues. Vomiting and purging now assert themselves, and the matters vomited and those passed from the bowels may be luminous in the dark, owing to the presence of free phosphorus. The vomit is at first made up of food, then mucus, then bile, then perhaps blood. All the symptoms of a mild gastro-enteritis may develop, but it is to be noted that constipation of an obstinate type may replace the purging. Very soon the liver increases in size, and gives rise to general hypochondriac pain and tenderness, as well as local swelling. At the end of twenty-four hours, or perhaps after the second day, a cessation in the symptoms occurs, and, if the physician be not on his guard, this will lead him to a hopeful prognosis. In the course of a few hours jaundice begins in the conjunctiva, and then extends over the entire body. With the onset of jaundice the vomiting and pain return with renewed vigor. The matters vomited are often the color of "coffee-grounds," due to exuded and altered blood. The bowels are absolutely confined, or the few hard masses passed are white and clay-like, because of the absence of biliary coloring-matter. There is no bile in the vomit in this stage, because the hepatic ducts have been closed by the inflammation set up in the liver. After this nervous symptoms ensue. Muscular twitchings, headache, vertigo, wild delirium, erotic convulsions, and finally unconsciousness and death occur. Sometimes the convulsions occur just before dissolution. Even if the patient survive the acute stage, he generally dies of the changes produced in his vital organs, which consist in widespread fatty degeneration, even in the acute stages. Atrophy of the liver, destruction of the gastric tubules, pancreatic involvement, and kidney degenerations aid in producing the ultimately fatal result.

During the course of poisoning by phosphorus the urine is scanty and perhaps albuminous, and is peculiar because of the unusual substances found in it. The most unusual of these is sarco-lactic acid, which results from the breaking-down of muscular tissues. Leucin and tyrosin are also found, and tube-casts, with fatty globules in them, are seen. Free fat-globules may also occur. Bile acids and bile coloring-matter are found in large amount, and the urine is generally dark colored for this reason. As phosphorus is eliminated as hypophosphoric acid, this substance is also present.

The symptoms may so closely resemble those of acute yellow atrophy of the liver as to make a differential diagnosis impossible, unless some evidence of the presence of phosphorus is obtainable.

Vomiting of a nervous type is a common complication of *exophthalmic goitre,* and when it occurs sometimes develops into a dangerously severe symptom, owing to its constancy, violence, and resistance to treatment. Oftentimes serous diarrhœa is associated with it, and these two associated symptoms should not mislead the physician into a diagnosis of cholera morbus nor of jaundice, for icterus often comes on.

Ménière's disease is associated with vomiting, the contents of the stomach being expelled after the attack of vertigo and tinnitus aurium.

The *affections of the liver* which sometimes result in vomiting are chiefly catarrhal and obstructive jaundice, hepatitis, hepatic abscess, and pylephlebitis. The rapid development of jaundice, hepatic tenderness, and swelling, or a history of violent hepatic pain (colic), renders the diagnosis possible in the case of jaundice. (See chapter on Skin.) Hepatitis—that is, hepatic abscess—is often accompanied by or produces vomiting which is apt to be very obstinate. The swelling of the liver, the tenderness in the hypochondrium on palpation, the pain in the hepatic region, often referred to the neighborhood of the right shoulder, and the febrile movement, which is intermittent, sweeping up to 104° or 105°, then down to normal, are the chief characteristic symptoms. (See chapter on Abdomen.) Vomiting accompanied by a similar train of symptoms also occurs in cases suffering from pylephlebitis.

Violent vomiting associated with great pain in the loin, radiating down into the testicle, or inside of the thigh, indicates the presence of a *renal calculus*, either in the pelvis of the kidney or in the ureter.

Hæmoglobinuria is sometimes accompanied by vomiting. The attacks are paroxysmal, and are often ushered in by persistent yawning, with pain in the limbs, headache, nausea, and vomiting, followed by moderate fever, which is preceded or accompanied by a chill. Pain may be felt in the liver, but, more pathognomonic than all, the urine is soon found to be dark, brownish-red, or even black, owing to the presence in it of hæmoglobin.

Vomiting is a frequent coincident symptom of headache, because in many cases the headache depends for its existence upon a disordered stomach or disordered bowels; but it also appears as a characteristic symptom of a condition in which the stomach is primarily not at fault, namely, in *migraine* or *hemicrania*, in which, in addition to violent pain in the head on one side, we have hemianopsia, scotomata, and sometimes great pallor or flushing of the face. (See Pain.) Usually the vomiting follows the pain.

In *acute yellow atrophy of the liver* vomiting is a constant symptom, with jaundice, violent headache, and finally convulsions and subcutaneous ecchymoses. (See chapter on Convulsions.)

When vomiting occurs in *yellow fever*, the presence of an epidemic, the suffusion of the eyes, the headache, the black character of the vomit, the slow pulse, scanty urine, and prostration, all point to the cause of the symptom.

In *Addison's disease* vomiting is often present, and may be so severe as to prove beyond control. The pigmentation of the skin (see Skin), the mental and physical feebleness, headache, vertigo, and anæmia without loss of weight in many cases, all aid in the diagnosis.

Vomiting is a frequent symptom in some cases of *phthisis*, particularly if laryngeal tuberculosis is present. It also occurs as a result of swallowing the sputum instead of expectorating it, and very commonly excessive cough produces vomiting, especially if the cough follows closely after a meal.

Closely associated with the vomiting due to these causes is that occurring in cases of *pulmonary gangrene.*

In children suffering from *whooping-cough* vomiting often takes place at the close of the paroxysm, and is due to the spasmodic movements of the chest and diaphragm.

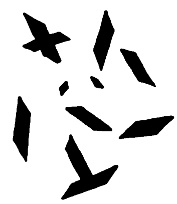
Finally, it is not to be forgotten that vomiting often ushers in any one of the *eruptive diseases,* such as the fevers, syphilis in its early secondary stages, and erysipelas.

Under the name of merycismus cases of *voluntary regurgitation* of food have been reported chiefly outside of the United States. The patients have been nervous or hysterical persons.

THE VOMIT.

Aside from the diagnostic significance of the act of vomiting, the physician should remember that the vomit itself may give him information as to the condition of his patient.

FIG. 233.



Hæmin crystals.

Under the head of gastric dilatation we have spoken of the significance of vomiting large amounts of liquid and undigested food, so that it is not necessary to speak of this point here; but it is well to remember that small amounts of vomited material often possess considerable diagnostic importance. Thus, in the case of drunkards it is by no means rare for the patient to vomit in the morning small amounts of watery and sour material, and in the severe retching of cerebral disease or uræmia very little material is gotten rid of, and this may be ammoniacal. In cases in which small quantities of exceedingly sour, clear liquid are vomited, we often find that the attack is due to migraine or nervous headache. If watery liquid and mucus are vomited, there is probably gastric catarrh. The vomiting of bile is usually only seen when repeated retching has drawn this secretion into the stomach. The liquid may be either golden yellow or greenish in hue. Somewhat like this vomit is that seen in peritonitis, in which disease grass-green material is often expelled. Similar material is also vomited in cases of intestinal obstruction before stercoraceous vomiting comes on.

The significance of bloody vomit has already been described. It only remains to name the test for hæmin, which, if present, shows that the color of the ejecta is really due to blood. Some of the vomited material is filtered, and a little of the filtrate is evaporated on a watch-glass. This dried material is now scraped off the glass, mixed with a trace of finely powdered common salt, and placed on a glass slide. The powder is now covered by a cover-glass and one or two drops of glacial acetic acid allowed to flow under the cover-glass. This is then dried by exposing it to gentle heat, and after the drying is absolute a drop or two of pure water is touched to the edge of the cover-glass. Very minute crystals of hæmin are now seen with the aid of the microscope (see Fig. 233).

The vomit of intestinal obstruction is sometimes fecal in odor for obvious reasons. If odorous poisons have been taken, the vomit smells of the poison; and if there be phosphorus present, the vomit not only smells of it, but in addition may be luminous in the dark.

CHAPTER VI.

COUGH AND EXPECTORATION.

The varieties of and diagnostic significance of cough—The causes of cough—The sputum—Its pathological significance.

THE significance of cough as a symptom is very important, and, though it may arise from many causes, in the majority of instances it points to disease in the chest, in the trachea or the larynx, in the pharynx or in the nose. Rarely it is a purely nervous trick, and equally rarely it arises from irritation in the stomach ("stomach-cough," so called). A cough is said to be dry and hacking when it fails to bring up into the throat or mouth any secretion, or when it is short and sharp. Often such a cough is paroxysmal; in other cases it consists in single but fairly frequently repeated, short, and forcible expiratory efforts, as if the patient was trying to clear his throat. A loose cough is nearly always paroxysmal; that is, it occurs "in spells," and at nearly every paroxysm results in the raising of some mucus. The first variety of cough is that seen in the early stages of phthisis pulmonalis, acute bronchitis, or pneumonia, before any exudation has taken place; in the early part of a paroxysm of asthma; in the early portion of an attack of whooping-cough and when the cough arises from irritation in the upper air-passages, whether this be due to the inhalation of dust or the presence of some growth, as a laryngeal papilloma. The loose variety of cough is seen in the later stages of acute bronchitis, pneumonia, asthma, whooping-cough, and in cases of emphysema with bronchiectasis, and in the stage of pulmonary tuberculosis associated with the breaking down of lung-tissue, the formation of cavity, and the development of bronchitis with it, and in gangrene of the lung.

There are two peculiar forms of cough to be mentioned, namely, the so-called barking, brassy, laryngeal cough, which we hear most typically in false or spasmodic croup, and the cough of whooping-cough, which is, as its name implies, the most typical which we meet with. Suddenly the child begins to give a series of quick, sharp coughs, which become more and more rapid until the chest is emptied of air. In the early stages of the disease this is all that occurs, and unimpeded inspiration ensues; but later the cough no sooner ceases from exhaustion of the lungs of air than with the attempt of deep inspiration the glottis closes spasmodically, and the air is sucked through the chink with a whooping sound. The flushed or cyanotic face of the child, associated with these paroxysmal attacks, renders the diagnosis easy.

There is nothing distinctive in the cough of early stages of pulmonary inflammation, whether it be bronchial or vesicular, although, if the bronchitis be very intense or if the pulmonary inflammation also affect the pleura, the cough may be partly smothered or suppressed by the patient, who endeavors to control or stop it in order to escape the pain it causes. To this end he sits or lies in bed, endeavors to fix the muscles of his chest so that they will not respond to the reflex cough-impulse, and shuts his lips to hold his breath in, although very often the reflex irritation overcomes his will-power and the cough bursts through his compressed lips with an expression of pain. Such a cough is always indicative of pain.

In all forms of dry cough there is now and again a small plug of mucus expelled from some part of the respiratory mucous membrane. Such coughs possess no value to the patient, being merely a sign of reflex irritation; but a loose cough, unless it is very excessive, is of the greatest possible use to the patient, for it is an effort on the part of nature to rid the lungs of abnormal exudations or secretions. For this reason this symptom is not to be removed completely in cases of resolving pneumonia, pulmonary tuberculosis, or bronchiectasis with excessive secretion, since, if drugs are given which stop the cough, the lungs are speedily filled with the secretion; and in the case of tuberculosis or gangrene or muco-purulent bronchitis septic absorption with systemic infection results. Similar good results are reached by the cough heard in cases of pulmonary abscess, and when an empyema has broken into a bronchial tube. When the patient complains of chronic cough, which is worse in, or confined entirely to, the morning hours, and tells us that the cough finally causes the discharge of much secretion, and that this is followed by freedom from cough for many hours, the case may be one of tuberculosis with cavity, pulmonary abscess, empyema which has ruptured into a bronchus, or sacculated bronchiectasis. Such coughs come on in paroxysms whenever the lung must be relieved, and the length of paroxysm depends upon the looseness of the secretion and its situation in the lung. Thus, if the secretion be in the larger bronchial tubes, it is easily expelled; whereas if it be in smaller bronchioles, or in the vesicles, or at the bottom of a cavity, great and frequently repeated effort will be required before the liquid can be raised into the mouth for expectoration.

The presence of an obstinate cough due to bronchitis, which resists all ordinary treatment, should lead the physician strongly to suspect that one of two ailments is present, namely, undiscovered tuberculosis, or, if there is puffiness under the eyes, Bright's disease.

Cough brought on by changing the position of the patient often arises because of the alteration in position of a pleural effusion. Violent and constant cough often comes on during aspiration of the chest for pleural effusion.

The cough of acute laryngitis may be quite severe, and occurs in short, sharp barks of a harsh or brassy character (like spasmodic cough) which is so typical as to be called a laryngeal cough. The association with this cough of partial or complete loss of voice and pain in the larynx, with a history of exposure to cold and dust, or the excessive use of the larynx in speech or singing, renders the diagnosis clear, even if the laryngoscope is not used to discover congestion and inflammation of the laryngeal mucous membrane. In the false croup of children, which is always associated with laryngeal irritation, the barking, ringing cough is so characteristic as to render a diagnosis possible as soon as the sound is heard, and with it there is dyspnœa due to obstruction to breathing.

The cough of the laryngeal phthisis is not so typically brassy and ringing as that of acute laryngitis, but the presence of pain in the larynx, hoarseness, and persistent laryngeal dryness and difficulty should lead to a search for tuberculosis by the laryngoscope, and an examination of the chest for physical signs of trouble in the lungs and of the sputum for tubercle bacilli.

Sometimes cough of a laryngeal character is due to an aneurism pressing upon the larynx. In other cases the cough depends not upon the pressure of an aneurism, but upon the pressure produced by carcinoma of the œsophagus, by a mediastinal tumor, or is due to the inhalation of irritant dusts or vapors. This cough, laryngeal or bronchial, is often present in girls who work in carpet factories, in the air of which there are immense quantities of fine particles of wool. Again, it is seen in knife-grinders, needle-workers, coal-miners, and in workers in arsenical and lead pigments.

Sometimes in paralysis of the pharyngeal muscles (glosso-labio-pharyngeal paralysis) cough is produced by the slow passage of food, which may in fact enter the larynx.

A night or evening cough is very commonly seen in cases of respiratory catarrh or more grave disease. It is often absent all day, only to return in the evening in cases of laryngitis and in phthisis; and, in those cases in which it follows getting into bed, it is due to chilling of the skin by the cold sheets, which results in congestion of the inflamed mucous membrane.

Quite frequently children suffering from chronically enlarged tonsils suffer from cough on going to sleep, especially if the uvula is relaxed or elongated. The cause of this cough is that in the relaxation of sleep the tonsils touch one another or tickle the uvula. As soon as the child wakes muscular contraction separates the approximating surfaces and the cough soon ceases.

If this cause of cough cannot be eliminated we must look further for its origin. Not infrequently hypertrophy of the mucous membrane over the turbinated bones, so that it presses on the nasal septum, may cause cough, and irritation of the inferior and middle turbinated bodies and the septum opposite the inferior turbinated body may cause reflex cough. So, too, enlargement of the pharyngeal tonsil may cause this symptom, as may also elongation of the uvula. When chronic enlargement of the tonsils, with follicular accumulations, is present, cough frequently results.

Sometimes in hysteria a peculiar barking cough is produced by pinching the skin of the neck or elsewhere in the body, and this symptom may also come on in paroxysms independent of such causes in neurotic children of both sexes about the time of puberty.

In regard to coughs in general, it can be said that in the absence of the early stages of acute inflammation of the respiratory apparatus a dry, hacking cough is either nervous or is due to reflex irritation in the ear or stomach, or to some hyperæsthetic spot in the nasal, pharyngeal, or laryngeal mucous membrane; whereas a loose cough may arise both in adults and children from congestion and engorgement of the lingual tonsil. Such a cough is frequent, dry, and paroxysmal, and seems to arise from a sticking or pricking sensation in the throat; whereas a loose and prolonged cough depends upon causes of greater gravity further down in the respiratory organs. Generally, if the stomach is at fault, a low grade of pharyngitis will be found. The physician should also remember that valvular disease of the heart, by the secondary changes which it causes in the lungs, may produce cough, and Sansom states that cough was present in 45 per cent. of the cases of heart disease taken by him as illustrative.

There is another variety of cough seen in persons who have paralysis of the diaphragm. The cough under these circumstances is produced for the purpose of expelling air from the chest, and is seen in persons with paralysis of the diaphragm from the removal of large ovarian tumors, which have pressed upon it and stretched it and the chest-walls to which it is attached, so that when the pressure is removed the muscle is too slack to contract. It is heard in injuries to the phrenic nerve and to the spinal cord.

The cessation of cough in advanced phthisis, suffocative bronchitis, or the bronchorrhœa with bronchiectasis of old persons, or in severe pneumonia, indicates exhaustion, collapse, or approaching unconsciousness, and is a bad sign.

The Sputum.

Macroscopic Examination. A careful examination of the materials expectorated by the patient, or, in other words, of the sputum, is of the utmost importance in all cases of disease of the respiratory tract, whether the abnormal process be primary or secondary. The methods which we resort to in examining sputum are macroscopic, or those of the naked eye, and microscopic. Of these, the microscopic are by far the most important. Sputum varies greatly in its general character on ordinary examination, sometimes being very fluid and even watery in consistency, and sometimes thick or tenacious. In some instances it is clear and glairy-looking, resembling somewhat partly beaten white of egg; in others it is yellow and opaque. Placed on a clean linen cloth, the sputum may evaporate to almost nothing, or leave a heavy muco-purulent deposit after all moisture is gone.

The naked-eye appearances of sputum are, however, quite characteristic in several conditions. Thus, in the later stages of acute bronchitis the sputum is apt to be thick and yellowish, and to contain lumps of half-inspissated mucus. In croupous pneumonia it is rusty in color, is peculiarly free from watery ingredients, and is gelatinous to such an extent that it adheres to the spit-cup, so that when this vessel is well filled its contents do not readily fall out even when the cup is tipped upside down. The brightness of the blood in the sputum in cases of pneumonia is also a guide to prognosis. Thus Sir William Jenner said, "the less the weight for a given height, the more red blood in the sputum, the better the chance for the patient." Later on the sputum may be less adhesive after resolution is well advanced. If the pneumonia is prolonged, the sputum has such a dark color that it is often called "prune-juice sputum." A third variety is that seen in pulmonary hemorrhage or hæmoptysis. In this condition, after having, perhaps for a short time, a salty taste in the mouth, the patient suddenly brings up, with or without much preceding cough, a gush of nearly pure blood or blood freely mixed with ordinary sputum. The blood is bright red, not dark or prune-juice in appearance, and the liquid is frothy, while the cough, which is always present after the hemorrhage has occurred, is suppressed and resisted by the patient, who fears further bleeding. This hæmoptysis may be caused, first, by pulmonary tuberculosis; second, by valvular cardiac disease, generally involving the mitral valves, and resulting in pulmonary infarction; third, by aortic aneurism; fourth, in persons suffering from severe purpura; fifth, from persons suffering from hæmophilia; sixth, from vicarious menstruation; and, seventh, in rare cases of hemorrhagic smallpox.

Bloody sputum must be separated from bloody vomit due to gastric hemorrhage arising from ulcer or cancer (see Vomiting). This can be done by the cough, by the frothy character of the expectoration, by the presence of physical signs in the lungs, and by the history of pulmonary disease. It may, however, be confused with slight hemorrhage from a dilated and ruptured vessel on the posterior pharyngeal wall, in which case, after a little coughing, there may be expelled on a handkerchief a little blood-tinged saliva. Examination of the throat will usually reveal the ruptured vessel or other vessels dilated, but still intact. For a number of days after an attack of hæmoptysis there may be expelled in the sputum dark clots of blood. So-called "currant-jelly" clots are expelled by coughing in many cases of malignant growths of the lungs.

Other causes of blood-streaked sputum are aortic aneurism with leakage by oozing into a bronchus or the trachea; and particularly in children do we see streaks of blood in the sputum if there be present pulmonary gangrene.

Care should always be taken to discover whether the materials spit up are really tinged with blood, for they may be colored by some dye-stuffs or the blood of some animal for the purposes of deception.

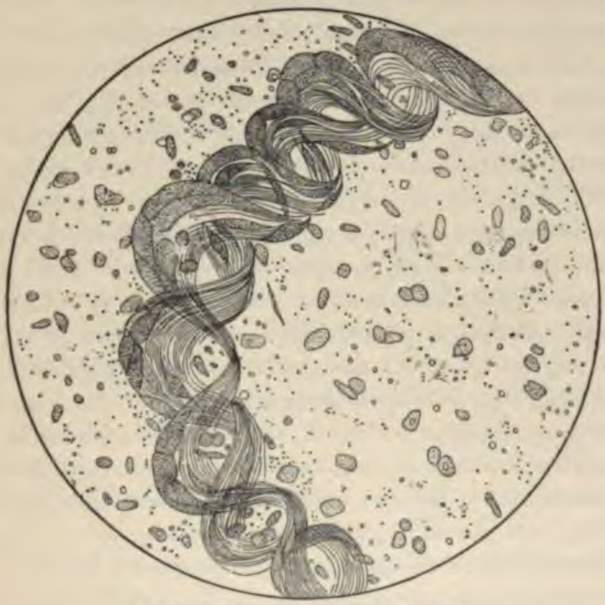
Finally, it is well to remember that a reddish-brown or brick-dust looking sputum is sometimes coughed up in cases of hepatic abscess communicating with the lung; and the sudden expectoration of a brownish, purulent-looking sputum by a person who has been a sufferer from dysentery should cause the physician to examine the sputum for the amœba coli, in order to discover if the case is one of pulmonary abscess secondary to amœbic dysentery. Symptoms of hepatic abscess may also be present. This has been called "anchovy-sauce" sputum.

In addition to the sputum already described we sometimes see a peculiar semi-liquid sputum in cases of pulmonary phthisis, in which the sputum promptly separates into two layers on standing, the upper one being light and flocculent, unless there is a well-marked bronchial catarrh present, when it may be markedly muco-purulent, or, if a large cavity is present, its purulent character may be even more marked. To this list may be added several others, namely, the purulent sputum of pulmonary abscess or empyema, of mediastinal abscess opening into a bronchus, subphrenic abscess, hepatic abscess, pronounced bronchiectasis, and that from a large tubercular cavity in the lung. Of these the more common are bronchiectasis, tubercular cavity, and empyema breaking into a bronchus. In the first of these the cough is paroxysmal, and after it has been kept up for some time a gush of purulent sputum is suddenly brought up into the mouth, and the accumulation of pus is removed for a time. In the other the sputum is very fluid, and is so free that its expectoration rapidly fills the spit-cup, provided that the patient is strong enough to bring it up. A very liquid watery sputum is seen in pulmonary œdema, particularly that seen in cases of Bright's disease or heart-failure, and in some of the pulmonary forms of epidemic influenza.

Sputum which, on standing, separates into three layers, the top one frothy and dirty looking, the next clear and filled with shreds, and the lowest consisting of a sediment of pus and broken-down-looking materials, is seen in cases of pulmonary gangrene, and if the sputum when placed in a vessel containing water sinks to the bottom in disk-like masses or globules the disease may be tuberculosis.

The sputum expelled by an asthmatic at the time of the attack also has characteristics not so easily seen at a glance, but nevertheless demonstrable by the naked eye. Small pearls or plugs of mucus of the size of a sago-pearl are seen in the sputum, and if these are placed on a plate of glass under which is a black surface, and then teased out, they will be found to be rolled-up fibres, which when unrolled are found to be in the forms of curls or spirals. These are sometimes called Curschmann's spirals (Fig. 234), and they are rarely seen in several other conditions than asthma, namely, in chronic pulmonary tuberculosis and croupous pneumonia. Through the central core of the curl runs a bright and refractive filament, which is waxy, and is probably not an entity, but an optical effect.

FIG. 234.



Curschmann's spirals. (VON NOORDEN.)

Between the spiral fibres can be seen in many cases slight, bluish, octahedral crystals varying greatly in size, sometimes requiring a high-power lens to distinguish them. They are said by Salkowski to be composed of a mucous substance, but others believe them to be oxalate of lime, a phosphate of an unknown base, or ethylenimin. These are called Charcot-Leyden crystals, and are also seen in the sputum of chronic croupous pneumonia, chronic pulmonary tuberculosis, and in acute bronchitis.

Sometimes, in cases of diphtheria, casts of the larynx and upper bronchial tubes are expelled by coughing. Small casts are also seen in the sputum of that rare affection, fibrinous bronchitis. Sometimes these casts consist of a perfect mould of several branching bronchial tubes and bronchioles, and they may be white, yellowish, or even pinkish in color from bloody exudation. Sometimes they are only visible to the naked eye if placed in water and shaken, when what has appeared to be a roll of mucus spreads out into the characteristic shape of the tubes from which it comes. Casts of the finer tubes can sometimes be found in the sputum of cases of croupous pneumonia.

Microscopic Examination. The examination of the sputum by the aid of the microscope should be made with care. A portion of the sedimentary part of any sample may be carefully separated from the rest by means of a pipette; but to facilitate the examination of sputum for tubercle bacilli when but few exist, and for the rapid and thorough examination of the sputum for elastic fibres, which has been heretofore a tedious and complicated process, and for the discovery of Charcot-Leyden crystals and fibrinous coagula, the centrifuge is a much better apparatus. (See chapters on Urine and Blood.) The tubes used for the precipitation of the sputum are 50 mm. long, with 2½ mm. lumen, and are fitted into the hæmatocrit frame.

A small quantity of sputum is placed in a clean porcelain dish and actively stirred for a few minutes with a glass rod until it becomes thin and free from lumps and apparently homogeneous. Without any dilution whatever the sputum is drawn into the sputum-tubes by means of a medicine-dropper connected with a small rubber tube. The two precipitating tubes, filled with sputum in this way, are placed in the hæmatocrit frame and revolved for at least three minutes, making about fifteen thousand revolutions. The solid portions of the sputum collect in the distal extremity of the tubes. A small portion of the sediment is placed on a slide and examined microscopically for elastic fibres, Charcot-Leyden crystals, etc. The sediment from the second tube can be stained for tubercle bacilli or other micro-organisms as desired.

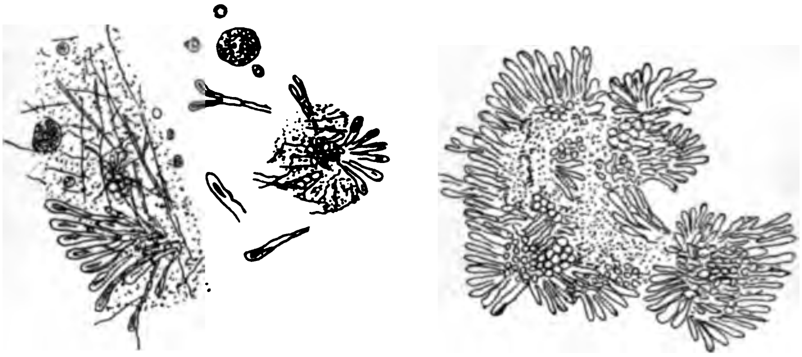
When the centrifuge is not used and a small particle of sputum is placed upon a glass slide under a cover-glass and gently pressed, there will be seen, if the sputum be mucous or muco-purulent, threads of mucus and mucous corpuscles with white blood-corpuscles, which are particularly numerous if the sputum be purulent. These latter are granular, fatty, and sometimes pigmented by soot or other substances which have been inhaled. Epithelial cells derived from the respiratory passages are also found in large numbers, often broken down and fissured, granular, and generally a nucleus can be distinguished in their centre. Of far more importance than these, however, are the particles of elastic fibre or elastic threads, which, if present, show positively that a breaking-down process is going on in the lung, or more rarely in the bronchial tubes. These are usually seen in the sputum of advanced or rapidly progressing tuberculosis of the lung and in that of abscess and gangrene of the lung. If there is doubt as to their presence because they are sparse, we obtain them by the following process: boil equal parts of the suspected sputum and a 10 per cent. solution of caustic potash, and dilute the jelly-like mass which results with water. After this has stood for twenty-four hours the elastic fibres may be found in the sediment as swollen threads, for which, however, small particles of food which may come from the mouth may be mistaken.

The appearance of fine, needle-like crystals of fatty (margaric) acid, which may be bent like a curved needle and often grouped in bunches, may indicate pulmonary gangrene or purulent bronchitis. They are found chiefly in the plugs or lumps which the patient expels in his sputum, but they possess no indicativeness of the pulmonary changes just named if follicular tonsillitis, either acute or chronic, is present, since the plugs derived from the follicles of the tonsils also contain similar crystals. Again, they are of no diagnostic value if found in stale, muco-purulent sputum, as they may form in this after it has been expectorated. The peculiar crystals called Charcot-Leyden crystals have already been described.

There are four remaining objects to be seen in the sputum of diseased persons, all of which are of great diagnostic importance when found. The first of these is very rare, namely, the eggs of the distoma pulmonum, which are found in the sputum. This parasite sometimes produces hæmoptysis without any physical signs of pulmonary change, and is rarely if ever seen in this country, but is common in Japan, Corea, Formosa, and China. The second is the evidence of echinococcus infection by the appearance of portions of the cysts or of the hooks of the scolices when the cyst bursts into a bronchus from the lung or adjacent structures. Such cases are rare in this country. The third condition, which is also very rare when involving the lung, is actinomycosis, in which infection we find radiated fungi or actinomyces in the sputum. This fungus appears as a number of club-shaped projections attached to a heterogeneous mass of irregular-looking material. (Fig. 235.)

The fourth and most important of all these finds in sputum is the tubercle bacillus, both from the point of diagnosis, prognosis, and treatment, and because the disease tuberculosis is so widely distributed in every class, in every part of the country, and is so constantly prevalent. The discovery of this bacillus in a sample of sputum which has not been exposed to the entrance of bacilli after it has been expectorated, is a positive sign of tuberculous infection of the lung unless there be tuberculous disease of the upper air-passages or mouth. While their presence gives positive evidence, their absence in a given sample of sputum is not negative evidence of an absolute character, for that particular specimen may be free from bacilli or they may have escaped the staining or the eye of the examiner.

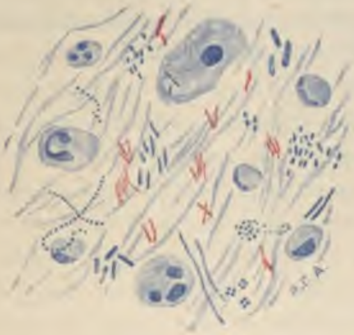
FIG. 235.



Actinomyces.

The method for examining sputum for tubercle bacilli is as follows : The specimen which is brought to the physician is poured into a shallow glass vessel having a blackened bottom, or into a glass saucer placed on a piece of blackened paper. If this sputum is now closely examined, it will be found to contain small, yellowish masses, one of which should be picked up by means of a spoon or platinum needle, freeing it as much as possible from the mucus surrounding it. A very small part of this mass is now placed on a cover-glass and well distributed over its surface by means of a needle or teasers, or it is spread by placing another cover-glass over the first and pressing them together with a to-and-fro movement. The quantity of sputum used must not be large enough to extend over the edges of the glass. This having been done, the glasses are now separated by a gliding movement, and the thin film of sputum covering each one is allowed to dry by exposure to the air, after which, being held by forceps, it is passed through the flame of an alcohol lamp to fix the coating. Care must be taken that too much heat is not used. Some carbol-fuchsin stain[1] is now placed in a watch-glass and the cover-glasses are immersed in it. As soon as this is done the cover-glass is held over a flame until the steam begins to rise from it, when it is withdrawn, then heated again until this process has been repeated several times. The cover-glass is now washed in water, and the film covering it will be found to have an evenly distributed red color. The glass is next placed for a moment in a 25 per cent. solution of nitric or sulphuric acid in water and gently moved about, to decolorize the deposit on the surface of the glass. As a result the albumin and cells on the glass are decolorized, but the bacilli are not. The glass is next washed in dilute alcohol (60 per cent.) to remove any free fuchsin. Should the decolorization be imperfect, so that the film still has a red color, it must be still further decolorized by returning the glass to the acid solution and then washed again in the dilute alcohol and water. The cover-glass is now placed in a solution of methylene-blue (saturated

FIG. 236.



Tuberculous sputum stained by Gabbett's method. Tubercle bacilli seen as red rods; all else is stained blue. (ABBOTT.)

[1] Carbol-fuchsin stain, or Ziehl's solution, is made by dissolving 1 gramme of fuchsin in 10 c.c. of alcohol, and adding 100 c.c. of a 5 per cent. solution of carbolic acid.

watery solution). The glass is then finally washed in water, and afterward examined under the microscope, or dried and mounted in Canada balsam for permanent preservation. The tubercle bacilli are distinguished by the fact that they retain the red color from the fuchsin solution, while other bacteria, having been decolorized by the acid solution, take the contrast-stain and appear blue.

Another method (Gabbett's modification of Fraenkel's) is perhaps more useful than that just given. It consists in staining, as already directed, with the carbol-fuchsin solution, and then placing the cover-glass in a second solution, which contains the acid for decolorizing and the contrast-stain. This latter solution is composed of 20 parts of nitric acid, 30 parts of alcohol, 50 parts of water, and sufficient methylene-blue to make a saturated solution, equalling above one or two parts in a hundred. The cover-glasses are left in this solution for a couple of minutes, and then washed in water. When placed under the microscope the tubercle bacilli will appear as red rods in strong contrast to the blue background.

CHAPTER VII.

PAIN.[1]

The kinds of pain—The significance of its locality—Colic.

It is manifest that it will be impossible for the author in this volume to enumerate all kinds of pain, both as to the situation, degree, and character. He can only mention those forms which possess considerable diagnostic importance. It should always be remembered that pain is the sign adopted by nature to notify the individual of some abnormal condition in his body, and in many instances pain is only developed when the attempt is made to move a part which from its condition had much better be allowed to rest.

Pain is generally described as darting or stabbing in character, when it occurs in a single or repeated paroxysm; as throbbing or pulsating, when it rises and falls in severity with the pulse-beat; as dull and aching, when it resembles the feeling associated with a bruise. Sometimes stabbing or darting pains are called lancinating, and the patient may state that the pain is tearing and rending in character. All pain is associated with direct or indirect irritation of nervous matter, but, if the nerve or nervous centres connected with a part be destroyed organically or arrested in function, we have a condition called anæsthesia. (See chapter on Skin.)

Not infrequently darting or stabbing pain is associated closely with actual disease of nervous tissue, which may be primary or caused by the pressure or irritation of a growth or some foreign body. Such pains are seen in neuralgias due to inflammation of the sheath of a nerve or its surroundings as it passes through a bony foramen; in neuralgia due to meningeal thickening; in the lightning or tearing pains of locomotor ataxia; from pressure upon the spinal nerves by spinal disease or in that caused by fractured bones. Again, we often meet with violent pain as the result of true neuritis, whether it be produced by infection, by injury, or by poisoning.

Throbbing pain is nearly always associated with the presence of congestion or local swelling in the part where the pain originates, and arises from the fact that the peripheral nerves are subjected to pressure, which is increased with each additional beat of the heart. Dull, aching pain is often produced by slow inflammatory or pathological processes in organs not well endowed with sensory nerves.

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[1] See also chapter on Headache.

There are two forms of pain yet to be considered which are peculiar in their character, namely, that due to a blow or injury to the testicle and ovary and that which occurs in cases of inflammation or morbid growth affecting bony tissues, particularly in the long bones.

There is another point in connection with the study of pain as a symptom of disease, namely, that pain is often referred to a point far away from the source of the symptom. Thus, the child with hip-disease complains of pain in the knee or in the ankle; the one with dorsal caries, of pain in the intercostal nerves anteriorly; and a stone in the kidney may cause violent pain in the penis or testicle.

The physician should always remember that the degree of pain must be determined in part by the expression of the face and movements of the body, for often these features of a case will show that the pain described so vividly in words is much exaggerated. The general systemic signs of pain are a tense pulse, if the pain be recent in onset and acute; a somewhat accelerated respiration unless the pleuræ or lungs are involved, when it may be retarded; dilatation of the pupil; more or less sweating, particularly on the forehead; faintness; and sometimes the passage of clear, limpid urine if the pain be abdominal.

The first kind of pain which will be discussed is the darting or paroxysmal pain in what may be called the *neuralgias.* These depend upon one of three causes, and, though they may occur in any nerve of the body, are most commonly seen in the nerves of the head; or in nervous women in the nerves of the pelvic organs and external genitals. The three causes are generally debility with anæmia, reflex irritation, and irritation of the nerve by poisons or by the presence of growths.

Violent neuralgia of the head is commonly seen in overworked or overdanced young women, who lack sufficient sleep and fresh air and who are anæmic. It also arises from the reflex irritation of a decayed tooth, or from inflamed or overstrained eyes, or from a diseased ear, so that an examination of any one of these parts may reveal the cause of an obstinate neuralgic pain. Similarly we see cases of neuralgia, particularly of the supraorbital nerve, which are due to chronic poisoning by one of the metallic poisons, such as lead and arsenic, and also as a result of malarial infection (brow ague). If the neuralgic pain be due to neuritis, it will not only be typical of neuralgia, but along the track of the nerve marked tenderness will be developed on pressure, and rarely an eruption will appear on the skin, as a herpes zoster. Pure neuralgia, on the other hand, is often relieved by pressure upon the nerve involved.

When the fifth cranial nerve is affected by neuralgia, we find that if the upper branch is involved the pain is felt in the forehead, the eyebrow, and the eyeball, the conjunctiva often becoming injected. If the pain be in the upper lip, the posterior nares, and the cheek, the infraorbital or second branch is affected; while if the pain is in the lower jaw and chin, the third division of the fifth nerve is involved.

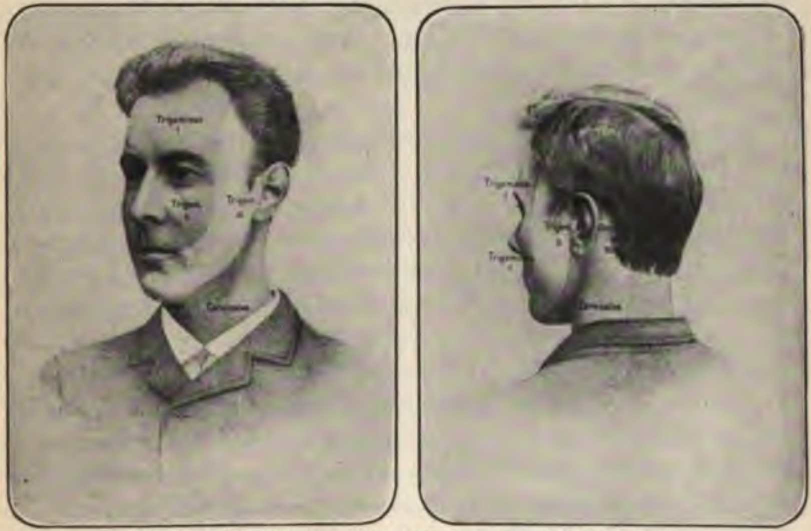


Fig. 237. Fig. 238.

Showing the distribution of the three branches of the fifth nerve.

A peculiar form of neuralgic pain coming on in attacks or paroxysms of great severity is *migraine* or *megrim*, in which the pain is usually confined to one side of the head, associated with great tenderness of the scalp, and may be preceded in many cases by disorders of vision, such as hemianopsia or dimness of visual perception. Associated with this pain at its zenith we frequently see vomiting and retching, faintness, with sweating localized in the pain-area, or diffused, and great facial pallor. Pressure by the fingers upon the painful area often produces no more pain or even gives partial relief, but a light touch may cause increase in the pain. Rarely a somewhat similar condition to migraine, which is not unilateral but bilateral, is found in connection with rheumatism of the scalp. As migraine may be due to a rheumatic taint, and this last-named condition arises from the rheumatic diathesis, care in making a differential diagnosis is necessary. The pain of migraine is, however, unilateral, more severe, more transitory, and associated with the symptoms named, whereas in the rheumatic head-pain the history of rheumatic tendencies of a marked character, the diffuse pain, the increased soreness on exposure to cold or changes in the weather, aid in separating it from migraine. When syphilis or injury causes a periostitis of the skull, violent pain of a neuralgic character may be present, particularly at night; but the local symptoms are manifest, and when compared with the history make the diagnosis possible.

It is also necessary to separate the headache of *cerebral tumor* or *cerebral abscess* from neuralgia of the head. The pain of such a cerebral condition is constant; the headache is sometimes worse at night, sometimes in the daytime, and greatly increased by physical or mental effort. The danger of confusing the pain of neuralgia with that due to tumor is great unless the physician remembers that the constant pain of tumor may vary from slight headache to sharp paroxysms of pain. The occurrence of convulsions points strongly to tumor if associated with headache of this character, and, finally, the presence of tumor as a cause of headache and not ordinary neuralgia is decided upon by evidences of optic neuritis, vomiting, vertigo, and the development of focal symptoms of localized paralysis. (See chapters on Headache and on Vomiting, Convulsions and Spasms.)

The most common seat for neuralgic pain in the head, other than in the brow, is the occipital region, the posterior branch of the second cervical nerve or great occipital being the one most affected. (Fig. 238.) As this nerve supplies the occipital region and the posterior part of the parietal regions, these areas may be involved in the painful manifestations, and all these parts may be tender to the touch. Brushing the hair may be impossible, because of the pain produced by the brush touching the scalp. Occipital neuralgia is oftentimes bilateral. It may simply arise from cold or injury; but, if persistent and severe, caries of the cervical vertebra should be sought for as a possible cause.

Pain of a neuralgic or darting character in the neighborhood of the heart is found as the result of several causes, as a rule in the following order of frequency: 1. Pain with palpitation of the heart from the accumulation of flatus in the transverse colon just as it turns to descend. Many patients who come to the physician complaining of heart disease only suffer from this condition, due to fermentation in the large bowel. Again, the pain due to gastralgia, or, as it has been called, cardialgia, may be referred to the heart by the patient. 2. To intercostal neuralgia due to debility. In these cases a tender spot will often be found, one in the præcordium, another in the outer edge of the scapula, and a third on the vertebral column. These are sometimes called the "spots of Valleix." In other cases the pain will be due to spinal disease, anæmia, or the tight lacing of corsets. 3. To pseudo-angina. 4. To true angina pectoris. 5. To locomotor ataxia.

Pain of a character somewhat resembling true angina pectoris is also sometimes met with in patients who have that rare disease, *acute aortitis*. The pain is constant under the sternum, but it has terrible exacerbations, and a sensation of rending of the retro-sternal tissues. Death may occur in an attack. It is seen chiefly in gouty patients and in syphilitics. Very rarely it is seen in patients who have suffered from malarial poisoning.

Pain is felt much more commonly in disease of the aortic orifice than in lesions of the mitral orifice of the heart.

Pseudo-angina occurs most commonly in anæmic, nervous girls, or young women whose vessels are normal but who have hysterical tendencies. *True angina* occurs in those of middle age or advanced life or in young persons whose vessels are affected by syphilis. In the false form the sensation is as if the heart would burst. In the real form it feels as if the heart was squeezed tightly in a vise. In this form, too, the bloodvessels will usually be found hard and corded, atheromatous, and the blood-pressure high. The additional diagnostic points in favor of true angina pectoris are that the principal seat of pain is somewhat to the left of the lower and middle sternum, from which spot it may extend to the axilla and back and turn off to the occiput or extend down the arms to the hands, where a sensation of coldness may be felt. Sometimes even the abdominal organs and testicles seem to be affected. The patient is motionless, the face anxious and covered with a cold sweat, and respiration is shallow. The disease is usually seen in persons over forty years of age. The thoracic pain of locomotor ataxia is rarely felt in the præcordium, but commonly in the axilla, and it rarely radiates down the arm. The other symptoms of tabes dorsalis should be sought for in all doubtful cases. (See chapter on Legs and Feet.) True angina pectoris is far more rare in women than in men. Violent cardiac pain, exactly resembling that of true angina pectoris, is met with, according to Vergely, in diabetes mellitus.

Very severe pain, paroxysmal or constant, felt in the chest, may also be due to *aortic aneurism*, and, if so, will be found associated with pain shooting down the arm on the left side, dilatation of the pupil, unilateral sweating of the face and neck, and the physical signs described in the chapter on the Thorax.

Severe pain of a darting character felt in the chest, not due to angina or the causes just named, is nearly always an indication of one of four things: 1. Intercostal neuralgia, already named. 2. Pleuritis, with or without pneumonia. 3. Pericarditis, if it is felt in the præcordium. 4. A morbid growth in the chest, particularly a mediastinal tumor or enlarged bronchial glands.

Both *intercostal neuralgia* and *pleurisy* are associated with severe pain, increased by taking a deep breath, the pain occurring sometimes with inspiration and sometimes with expiration. They are to be separated from one another by the presence of cough, fever, and of a friction-sound in pleuritis, and by the fact that the entire side is more or less tender to the touch in this state. When the pain is constant and lasts for a long time, it may be due to a low-grade pleuritis, resulting from pulmonary tuberculosis, particularly of the apex of the lung, the morbid process affecting the pleura. *Pericarditis* is frequently caused by pneumonia, and is painful.

Pain felt at the right of the left scapula or between the shoulders is often due to gastric ulcer or dyspepsia.

The pain of mediastinal growth is due to pressure on nerve-trunks, and the diagnosis may be very difficult unless bulging and dulness on percussion are present.

Neuralgia of the *pelvic viscera* in women is frequently seen as the result of functional or organic disease. It may be ovarian, when it is very apt to occur with greatest severity half-way between the menstrual epochs or just before them. Sometimes the neuralgia may be present in the labia majora or in the perineum. It usually occurs simply as a sudden, darting pain, which does not last and, indeed, rarely continues more than a moment, although there is usually associated with it more or less constant uterine or ovarian tenderness. Care should be taken that these pains are not thought to be due to cancer or other severe organic lesion. Pain in the sacral region is often an indication of uterine or rectal disease. If higher up the back, it is often due to myalgia or lumbago; and lumbago, if not due to rheumatic tendencies, is often due to the colon being loaded with feces.

If the patient is a child, pain in the back should cause us to suspect *spinal caries, rickets,* or *scurvy.* If the former, any jar will greatly increase the pain; but if the child be placed over the knees, face downward, and the knees separated so that intervertebral pressure is removed, the pain disappears. Such a child if told to jump down from a stool will not obey, but will take care to slide off gradually and gently on to the floor, in order to avoid jarring the spine.

In scurvy the tenderness of the spine is usually diffuse, but it may mislead the physician into a diagnosis of spinal disease, when investigation of the gums will reveal scorbutic blisters or the diet will be found imperfect. (See chapter on Lower Extremities.)

When a patient suffers from violent pain, increased by motion, extending from the sciatic notch in the buttock down the posterior part of the thigh, even to the ankle or heel, the pain signifies an attack of *sciatic neuralgia* in an adult, or if it occurs in a child gives grave reason for suspecting *hip-disease.* If it is not sciatic neuralgia, it is due to sciatic neuritis, or, rarely, to a growth in the pelvis pressing upon the nerve before it emerges from the pelvis. The pain is fairly constant, generally worse at night, and becomes agonizing at times, even if the patient remains absolutely quiet and does not move the limb. The following points will, when pressed on, increase the pain if it be neuritis: the point of exit of the nerve from the pelvis, the lower part of the sacrum, the head of the fibula, and behind the malleolus on the outside of the ankle. If these points are found, combined with a history of exposure to cold, injury to the nerve, rheumatic tendencies, and a persistency and tendency to return, the diagnosis of sciatica is clear. If the pain be due to sciatic neuritis there may be found wasting in the muscles supplied by the nerve, and some anæsthesia of the skin, and herpetic eruptions may appear on the skin along the course of the nerves. There will be also a history of long duration, and the leg will be apt to feel numb and tense from effusions into the sheath of the nerve (See chapter on Skin and that on the Feet and Legs.) The heel and toes will be tender, but the inner aspect of the plantar surface will usually escape. Again, in sciatic neuritis, if the leg be extended, and then while in extension flexed at the hip till it is at a right angle with the trunk, pain will be felt at the sciatic notch. When the pain is a pure neuralgia, which is rare, it is not increased by moving the limb, there is little or no tenderness on pressure on the nerve-trunk, and the patient suffers from neuralgia of other nerves.

Sciatica is much more common in men than in women, which is the reverse of all other nerve pains of like character, and far more usual in middle or advanced age than in the young.

Double sciatic pain should arouse suspicion of *locomotor ataxia*, of malignant growth pressing on the spinal cord or on both nerves in the pelvis, the presence of lumbar abscess or of diabetes mellitus.

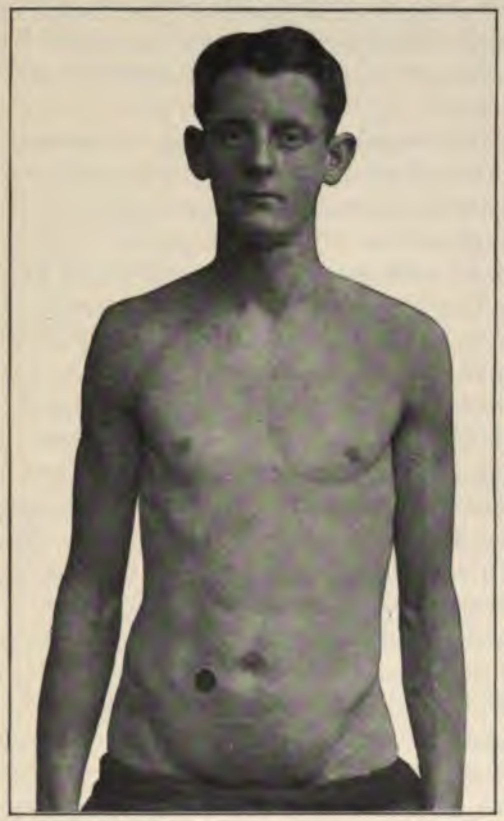
When there is a hysterical, painful joint at the knee or hip in a woman, care is necessary to discover that the pain is over the entire leg rather than in the course of the nerve. Care must also be taken that rheumatism of the muscles of the thigh be not taken for sciatica. This can be separated from sciatica by the diffuse character of the pain and tenderness and by the fact that in the rheumatic condition the slightest muscular movement causes pain all over the thigh. Sometimes a *malignant growth of the femur* may produce symptoms of sciatica, and the writer not long since had under his care a case of osteosarcoma of the femur which had been treated for sciatica for several months.

Finally, *renal calculus* may cause violent pain to be felt down the leg. (See below.)

It should also be remembered that malingerers, particularly soldiers desiring to shirk duty, often pretend to have sciatica.

ABDOMINAL PAIN. By far the most frequent form of abdominal pain is that due to flatus arising from indigestion. A peculiarity of this form of pain is the fact that it is rarely limited to one spot for any length of time, and is usually relieved by the passing of gas from the rectum. It may be associated with diarrhœa and a history of the ingestion of indigestible food. Further than this, percussion will elicit a tympanitic note in the area of greatest pain and distension, whereas, in intestinal obstruction due to tumor or intussusception, percussion may reveal dulness. So, too, a tumor or the presence of a floating kidney causing severe pain in the abdomen may be discovered by percussion or palpation, as may also an enlarged gall-bladder. The development of pain of great intensity in the right lower quadrant of the abdomen, associated with muscular rigidity of the abdominal wall, tenderness on palpation, a quick pulse, and vomiting in some instances indicates the development of *appendicitis*, a diagnosis which is aided, but not confirmed, if on examining the blood a distinct leucocytosis is present. It is worthy of note that the pain of appendicitis is often referred in its greatest intensity, and by intelligent patients, not to the neighborhood of the appendix, but to the epigastrium, or even to the region of the sigmoid flexure, and it is by careful palpation only that the area of greatest tender-

FIG. 239.



The black spot is at "McBurney's point."

ness is found in the appendicular region. Pain in the area of McBurney's point (Fig. 239) may be due to salpingitis, to phlebitis of the iliac vein, to renal colic, or even to wind colic.

The pain of colic due to flatulence can be separated from that of peritonitis by the fact that pressure gives comfort in the first instance and is unbearable in the latter disease.

By colic is meant a sudden, griping pain in the belly. It is met with chiefly in cases of stone in the ureter or gall-duct, but it is often due to irritating food, to wind, and to chronic lead-poisoning, to fecal impaction, intestinal obstruction, intestinal perforation, and ulcer of the bowels.

Generally diffused pain of a constant severe character felt all over the abdomen or localized at first in some particular spot, and greatly increased by pressure, should lead the physician to examine the case for a possible *peritonitis.* Nothing else causes such violent, diffuse pain unless it be acute enteritis. The well-flexed legs, the anxious face, the drawn upper lip, quick pulse, the exquisitely tender abdominal surface, the thirst, the moderate fever, and the rapid onset of collapse in fatal cases render the diagnosis easy.

In *pancreatitis* the pain is sudden in onset, violent, and usually felt chiefly in the left upper zone of the abdomen. The belly is distended, nausea and vomiting are present, and fever may be present also; delirium may come on, and death generally speedily ensues unless operative measures are resorted to. (See Abdomen.)

The onset of severe pain in the abdomen in the course of typhoid fever, while not necessarily an indication of *perforation of the bowel,* is nevertheless deserving of careful study. If it is due to perforation, as a rule it is violent enough to make the patient cry out if he be not stuporous, and the following symptoms will probably be present in whole or in part. The epigastrium and general abdominal wall will be fixed; the abdominal wall may be scaphoid or distended, but is probably hard. Percussion of the area of the liver may show obliteration of hepatic dulness, as a result of gas being between the wall and this organ, but while this is an important sign if present, its absence does not exclude perforation. Hiccough may be present, and perhaps vomiting. The facial expression is that of grave abdominal disorder. (See chapter on the Face.) An examination of the blood may show leucocytosis, and if such an increase in white cells is present, it is an important positive factor in confirming the diagnosis of perforation. An absence of leucocytosis not only does not negative the diagnosis of perforation, but it may confirm it, because if the shock be great leucocytosis may be prevented by the very intensity of the insult to the peritoneum.

Severe abdominal pain in typhoid fever may, however, be the result of wind colic, acute pleurisy, a distended bladder, cholecystitis, and impaction of feces.

Sometimes a patient who has *floating kidney* will suffer from severe renal pain, nausea, vomiting, and collapse, the symptoms simulating renal calculus. These attacks are known as Dietl's crises.

In *hepatic colic* the patient often, after some days of wretchedness and "biliousness," is seized by sudden and violent pain in the right hypochondrium, which is paroxysmal in character and causes quite as profound symptoms of general disorder as does the renal colic. Jaundice very commonly ensues in such cases with more or less rapidity, and fever of an irregular type is more often seen than in the renal form of colic. It is worthy of note that a gallstone when in the gall-bladder, or cystic duct rarely causes much pain, and its impaction in this duct does not cause jaundice. If it be in the hepatic duct jaundice is present, and pain is often marked, but is not so severe as if it be in the common duct.

If the stone be impacted in the common duct, then the most violent pain is present, and jaundice is marked as a rule, the stools being clay colored. If the symptoms develop suddenly, and pass away equally rapidly, the stone has probably escaped into the bowel, whereas if the stone becomes impacted in the papillæ of the duct, the jaundice and other symptoms become progressively worse. It is entirely possible for the same stone to slip back, and cause temporary relief, then to slip into place and precipitate another attack, or for another calculus to follow its predecessor, and cause a return of the symptoms. Pain is very indicative of stone. Thus in 80 cases of common duct obstruction pain was marked in 51, absent in 10, and unrecorded in 19, while in 79 cases due to other causes than stone pain was present in only 9.

If the stone is movable, there may be attacks of acute radiating pain with chills, and sweats due to absorption of toxic matter through the abraded mucous membrane. (See Charcot's Fever.)

Severe pain in the hepatic region may also be due to *acute cholecystitis.* There is usually local muscular rigidity, tenderness, vomiting, but the liver is not enlarged unless the cholecystitis is associated with cholangitis. This condition follows the infectious fevers, notably typhoid fever, and may be confused with appendicitis, which often causes cholangitis or abscess behind the liver.

The pain of *diffuse hepatitis* varies with the severity of the inflammation, and it may not be present unless the hypochondrium is palpated. In cholangitis it is rare unless suppuration is localized, but in *cholecystitis* of a chronic type the pain often comes on in paroxysms, and is associated with fever, enlargement of the gall-bladder, and great tenderness in its area on palpation. The liver itself is not enlarged. If suppuration is present, the symptoms of general sepsis may develop. The paroxysms of pain may vary like those of gallstone in the endeavor of the gall-bladder to extrude its thick mucopurulent contents. Care should be taken not to mistake a subphrenic abscess for cholecystitis.

When there is a history of violent colic situated near the hypochondrium, or in the epigastrium, not accompanied by jaundice and enlargement of the liver, the possibility of the cause being a *pancreatic calculus* is not to be forgotten. Such a condition is rarely met with it is true, and still more rarely do we find pancreatic stones in the feces, because they are friable and broken up in the feces before they are passed. Boas states that they are a frequent cause of so-called "neuralgia of the liver." It should not be forgotten that blocking of the common duct by gallstone also produces blocking of the pancreatic duct, and this in time may cause a complicating attack of acute pancreatic necrosis.

The pains just described are so severe and characteristic in their distribution that they cannot well be confused with those of *intestinal indigestion*, in which condition we have a history of the ingestion of bad food, a state of more or less flatulent distention of the entire belly, and, it may be, diarrhœa.

If the pain be due to *chronic lead-poisoning*, it centres about the umbilicus, and is of a twisting, knotty character, "as if the bowels, were being twisted around a stick." There is a history of exposure to lead in many cases, and a blue line on the gums can often be found.

If due to *fecal impaction*, there is a history of a continued tendency to constipation, with dry, hard stools, and a lump of hardened feces may perhaps be felt through the belly-wall.

If due to *intestinal obstruction*, the pain has no characteristic seat in any part of the abdomen, as a rule; but the general symptoms of this condition will be found present in the case. (See chapter on Vomiting and that on the Abdomen.)

Abdominal aneurism may cause severe pain by pressure on nerve trunks; and uterine and periuterine disease also cause, reflexly, epigastric pains.

Reference has already been made to the pain of *renal colic*. The characteristic symptoms of this condition are as follows: in renal colic the patient is suddenly seized with violent pain in the region of the kidney on one side, which passes down to the groin and even to the end of the penis. It is paroxysmal in character, and so excessively severe that it often produces sweating, vomiting, and even fainting. The condition is seen much more frequently in men than in women. The pain often suddenly subsides, leaving only a sense of soreness and tenderness in its track. The urine may be partly suppressed and bloody if the stone injures the ureter to any great extent. Pain simulating renal colic may be due to neuralgia or arise from several organic causes not connected with calculus. Thus, Habershon has stated that in valvular disease of the heart, particularly of the aortic valve, severe and colicky pains frequently radiate down into the right hypochondriac region, and Ralfe says into the renal region. Again, pain in this part may be due to aneurism of the aorta or of the mesenteric artery. Further, the accumulation of hard fecal matter in the colon may cause nephralgia. Finally, Ralfe calls attention to the renal pain felt generally in the right kidney by women who have exercised violently while wearing a tight corset, which has pressed upon the liver and kidney with great force on making a jump or sudden bend of the trunk. Sometimes a sudden "storm" of uric acid or an accumulation of oxalic acid in the kidney causes pain and tenderness.

The presence of constant pain and soreness in the abdomen on one side, frequent micturition, and occasionally the presence of blood in the urine, are symptoms of several renal states, such as pyelitis, renal calculus, a tumor in the renal pelvis, and tuberculosis of the kidney. The pain is chiefly in the region of the kidney and along the course of the ureter where it crosses the pelvic brim; and as it is often made more severe by standing and is of a bearing-down character, it may be thought in women to be uterine. Jolts or jarring of the body also aggravate it, as does the act of urination. Sometimes a good deal of pain is felt on the sound side.

If an examination of the urine reveals pus, this will indicate pyelitis; and if the pus contains tubercle-bacilli, tubercular pyelitis. The latter diagnosis is confirmed if tubercular disease is present in other organs, for renal tuberculosis is usually secondary. Small cheesy masses may be present in the urine in the latter state. Renal calculus will be attended by attacks of renal colic, which, as a rule, are more severe than those produced by the passage of masses of broken-down matter along the ureter. A history of having passed a stone at some previous time, and the discovery of blood-corpuscles in the urine are of importance in the diagnosis.

Pain in the abdomen of the darting neuralgic type, other than that due to gallstones, renal calculi, ordinary gastralgia, lead-poisoning, enteralgia, or malignant growth, may be due to *locomotor ataxia*. This possibility should never be forgotten, and the fact that the patient is an adult, complains of the most violent pain in the belly, and has no other abdominal signs, should make us search for the other signs of tabes dorsalis. Generally these attacks will be of a tearing, rending character; they may be beyond description in severity, and after they pass off the patient is left in a condition of nervous wreck. Sometimes the pain is in the stomach, sometimes in the bladder.

Grube has recently reported cases showing that diabetes may produce attacks of violent abdominal pain resembling the crises of an ataxia.

Neuralgic pain in the back and abdominal parietes very closely resembling, if severe, renal or hepatic colic is sometimes due to *gastralgia*, the paroxysms being very sudden in onset. In other instances the pain is in the epigastrium or hypogastrium, and is associated with so much tenderness on light pressure as to impress the careless with the belief that a gastric ulcer or cancer is the cause. Neuralgic spots can generally be isolated in such persons if the skin is carefully tested for its degrees of sensation, and these spots will be found to exist near where the nerves are given off from the spine or over the spine of the ilium.

Circumscribed abdominal pain of a constant character and generally of less severity than that just described, may be due to an abdominal tumor (see Abdomen), or to ulcer of the stomach or bowel.

In *dysmenorrhœa* the pain is sometimes so severe as to render the patient almost insane, but it differs from that of inflammation in that it is paroxysmal and that there is no real tenderness on pressure; and, again, the patient does not lie still, but tosses from side to side in the bed. The pain of *tumor* is usually produced by pressure on a nerve, and is increased by palpation in some cases, as is also that of ovaritis. In *cystitis* the pain is deep in the pelvis, radiating upward, and is associated with tenderness, vesical spasm, and tenesmus.

SOME ABDOMINAL CONDITIONS IN WHICH SUDDEN AND ACUTE PAIN FORMS A PROMINENT SYMPTOM.

Disease.	Mode of onset.	Character of pain.		Seat of pain.	Tenderness and pressure.
		In kind.	In intensity.		
Acute intestinal obstruction:					
a. Strangulation due to bands.	Very sudden.	More or less continuous.	Most intense, agonizing.	Epigastric or umbilical region.	Pressure at first relieves, afterward aggravates.
b. Acute intussusception.	Sudden to very sudden.	Paroxysmal.	Severe.	Epigastric or umbilical region.	Pressure at first relieves, afterward aggravates.
c. Acute volvulus.	Sudden.	Paroxysmal, but less than *b.*	Moderate.	Umbilical or over heart.	Pressure never causes pain.
d. Due to gall-stone or stricture.	Less sudden, gradual.	Paroxysmal later.	Moderate.	Often near seat of obstruction.	Tenderness over obstruction.
Appendicitis.	Very sudden.	Quite continuous.	Agonizing.	At first periumbilical, later about appendix.	Greatest over appendix or in left groin.
Acute peritonitis.	Very sudden.	Continuous.	Very severe	All over belly, but chiefly epigastric or umbilical.	Very tender everywhere except at very first.
Hepatic colic.	Sudden.	Aching, tearing, paroxysmal.	Agonizing.	Epigastric, radiating to between shoulders or to shoulder-blade.	Pressure at first relieves, then unbearable over gall-bladder.
Renal colic.	Sudden.	Aching, tearing, paroxysmal.	Agonizing.	Affected loin, passing down in front into testicle and bladder.	Tenderness over affected kidney.
Intestinal colic.	Sudden or gradual.	Paroxysmal.	Varies in severity.	Varies in position.	Relieved by pressure.

The pain of *fissure of the anus* is not at all proportionate to the lesion producing it. This pain may be atrocious and agonizing, and often is produced by a movement of the bowels, after which it lasts for some hours.

(For abdominal pain due to conditions associated with movements of the bowels, see chapter on Bowels and Feces.)

[1] Andrew's table, slightly modified.

Neuralgia of the toe and foot is not a very rare condition, and is sometimes called "Morton's painful toe," or *metatarsal neuralgia.* Severe pain at the base of the fourth toe comes on suddenly, and may radiate up the anterior aspect of the leg. Sometimes it is only dull, at other times it is so sharp and excruciating as to cause the patient to scream. It is separated from gout by the absence of any signs of inflammation in the part, by the fact that the big toe is not affected, and by the age and history of the patient. At times the base of the second toe is affected. Such a case will usually indicate that the patient has worn an ill-fitting boot.

Finally, in connection with this class of cases there should not be forgotten two others, namely, those in which, idiopathically or otherwise, growths form on nerves and cause pain; and, secondly, cases in which, the arm or leg having been amputated, a *neuroma* or catching of the end of the nerve in the scar causes violent pain in the lost part, according to the patient's sensation, because the perceptive centres have been trained to regard pain-impulses coming along this nerve as from its peripheral end. Thus a man whose leg may have been amputated years before will complain of severe pain in the amputated foot, although he knows it is off.

The pain of inflammation is often very severe and throbbing in character; but, if the nerves be affected in the area involved, it may be darting in its nature. We have already discussed pain due to inflammation in the tissues of the head and in the nerves. We now have to consider the indications of pain due to inflammation in the chest and abdomen.

Pain in the back is often very severe in the early stages of smallpox, of epidemic influenza, and in lumbago. One of the most misleading forms of pain of a severe character, involving the entire body, with fever, delirium, and a variable skin eruption and swelling of the joints, may in the early stage be thought to be smallpox or rheumatic fever, when in reality it is due to *dengue* or breakbone fever.

CHAPTER VIII.

TENDON-REFLEXES AND MUSCLE-TONE.

The knee-jerk and ankle-clonus—The arm-jerk—The significance of decreased and increased reflexes.

We have already had occasion, particularly in those chapters devoted to the Legs and Feet and the Arms and Hands, to speak of what are called the reflexes or "muscle-jerks." There is much discussion as to whether the muscular contractions produced by tapping the tendon attached to a muscle are the result of a reflex action, in which the spinal cord is directly involved, or whether it depends upon muscle irritability or tone. It is not necessary for purposes of diagnosis to enter into a discussion of this character, because the facts in our possession prove conclusively that variations in these muscle-jerks are of great diagnostic importance in diseases of the nervous system, whether they be true reflexes or not. The knee-jerk, or, as it has been called, the patellar reflex, is the diagnostic sign most frequently sought in studying nervous diseases associated with lesions in the spinal cord, because it is most easily developed.

This knee-jerk is best developed by directing the patient to sit in an ordinary chair and to cross the legs, the foot which touches the floor being kept near the rounds of the chair rather than well forward, and the upper leg being allowed to swing perfectly free over the under knee. By means of the finger-tips, the side of the hand, or a small rubber hammer, a blow is now struck upon the tendon of the quadriceps extensor midway between the patella and the insertion of the tendon below the knee, and, if the man is healthy, the hanging leg and foot are at once thrown forward, that is, the knee-jerk is developed.

If there be produced by the patient at the moment of the tap on his tendon a strong muscular effort, as by sudden clinching of the hands, this movement of the leg is exaggerated, or, to use the common term, the knee-jerk is "re-enforced."

The production of pain or a sensory stimulation will also increase the knee-jerk, as will also the stirring of the emotions as by lively music. On the other hand, enervating weather, loss of sleep, hunger, or anything which decreases general systemic and muscular tone decreases the knee-jerk.

The second most important test of the muscular tone is the test for what is called "ankle-clonus." If a healthy person while sitting rests the weight of the leg on the ball of the foot, the leg is very apt, in a short time, to begin to tremble, and finally to develop clonic movements. In disease these movements often develop as soon as the foot is placed in this position, and are at once developed if, while the leg is so placed, a sharp blow is struck above the knee or sudden pressure is made on the leg. In other cases the clonus is tested for by grasping the ankle with one hand and the toes with the other and bending the foot up toward the knee with a rather forcible push, which will stretch the muscles of the calf of the leg.

The biceps tendon is tested by placing the arm in semi-extension and tapping the tendon, when the forearm is still further flexed. In this connection mention should also be made of the cremasteric reflex, which is developed when the skin of the inside of the thigh is tickled, the cremaster muscle drawing up the testicle on that side. It is most marked in boys.

The chin-jerk is elicited by having the patient open his mouth slightly and then placing a flat object, like a paper-cutter or tongue-depressor, on the edges of the lower teeth. A sharp tap is now given to the flat object, when in health there will be a slight upward jerk of the chin.

The scapulo-humeral reflex of von Bechterew is elicited by tapping the skin on the spinal border of the scapula near its inferior angle. This usually causes a slight adduction and external rotation of the arm. It is exaggerated in neurasthenia, but its chief diagnostic value is in connection with lesions in the pyramidal columns above the cervical enlargement, in which state it is greatly altered in that we now find contraction of the posterior fibres of the trapezius, the deltoid, biceps, and forearm muscles, so that the shoulder is raised, the arm thrown from the side, the forearm flexed, and the fingers extended. Not infrequently the muscular response is so exaggerated that the muscles on the opposite side also respond. If the brachial plexus be diseased, the response on that side may be greater than on the side that is tapped.

Having learned how to test these muscle-jerks, we now turn to a consideration of what they mean when absent or abnormally increased.

A loss of knee-jerk is not characteristic of any disease unless this loss is associated with other symptoms which only need the discovery of this symptom to confirm the diagnosis. The nervous conditions in which we find the reflexes decreased or lost, taking the patella reflex as a type, are locomotor ataxia; peripheral neuritis; poliomyelitis, acute or chronic; transverse myelitis, if the disease involves the reflex arc; Friedreich's ataxia; diphtheritic paralysis; apoplexy, immediately after the shock; Landry's paralysis; spinal meningitis; spinal injuries, immediately after the accident; epilepsy, immediately after an attack; and chorea. We also find a total loss of reflexes in advanced diabetes mellitus and sometimes in diabetes insipidus.

By far the most common cause of the loss of the knee-jerk is locomotor ataxia, but any lesion involving the posterior columns of the cord or the posterior nerve-roots in the second, third, or fourth lumbar segment will produce the same results. Therefore, loss of knee-jerk is symptomatic of transverse myelitis of this region as well as of ataxia. Again, if the motor tract of the cord at these levels is diseased, the knee-jerk is lost, as, for example, in acute and chronic poliomyelitis or myelitis involving the motor part of the reflex arc; and, finally, peripheral neuritis, which blocks the pathway from the periphery to the cord, and from the cord to the muscles, also causes loss of knee-jerk.

If the cause of loss of knee-jerk be locomotor ataxia, we will probably find in addition to this symptom some difficulty in walking, particularly if the eyes are shut; a lack of steadiness if the feet are placed together when the patient stands with his eyes shut; Argyll-Robertson pupils, or a reaction to accommodation but not to light; attacks of severe pain in the body or limbs; and, it may be, laryngeal crises or spasms and atrophy of the optic nerve.

If the cause of loss of knee-jerk be neuritis, we will find tenderness on pressure along the nerve-trunks, diminished muscular tone, and some wasting; an absence of any disturbance of the bladder and no Argyll-Robertson pupil, laryngeal or other crises, nor optic atrophy.

Again, if the cause be acute poliomyelitis, there will be a history of sudden onset with fever, the limbs will be relaxed and flabby, the muscles will rapidly waste and become very feeble or paralyzed, and there will be no sensory symptoms whatever. The patient will usually be a child if the disease is acute. If the loss be due to transverse myelitis of the second, third, and fourth lumbar segments, the symptoms of paraplegia, paræsthesia, and anæsthesia, with atrophy of the muscles and loss of control of the bladder and rectum, will be present, and a girdle sensation may be marked.

In Friedreich's ataxia the history of heredity, the nystagmus, the early age of the patient, the absence of pupillary symptoms, the ataxic gait, and the loss of reflexes, are the facts which go to form our basis for a diagnosis. In the remaining diseases named the history of the case points to the cause of the loss of the knee-jerk very clearly.

The conditions in which we find the knee-jerk *increased* are apoplexy soon after the attack; disseminated sclerosis; cerebral palsy of childhood; paretic dementia (not constant); primary lateral sclerosis; amyotrophic lateral sclerosis; ataxic paraplegia; hysterical paraplegia; transverse myelitis if the lesion is above the reflex arc; epilepsy some minutes after the attack; unilateral lesions of the cord on the paralyzed side; injuries to the spinal cord, after the recovery from first shock; pressure on spinal cord above the reflex arc; hereditary cerebellar ataxia; sciatica; tetanus; rheumatoid arthritis; and neurasthenia.

The history of sudden paralysis and unconsciousness in a case of apoplexy with stertorous breathing, followed by loss of the knee-jerk, and then its return in an exaggerated manner, make the diagnosis clear unless the attack be one of the apoplectiform attacks of disseminated sclerosis, in which case there will be present a history of the intention-tremor, nystagmus, and the syllabic speech, so that though the knee-jerk is exaggerated in both diseases the diagnosis can be readily made. In the cerebral palsy of childhood the age of the patient, the contractures and gait, with the history, decide the diagnosis. In lateral sclerosis the spastic rigidity, excessive exaggeration of the knee-jerks, absence of sensory disturbances, and ocular symptoms, all render the diagnosis possible. Similar exaggeration is also seen in amyotrophic lateral sclerosis, in which disease there is wasting of the muscles, particularly of the hand. In both these ailments the exaggeration of the knee-jerk is due to disease of the lateral pyramidal tracts, which block the inhibitory fibres from the higher centres. For similar reasons we find exaggerated knee-jerk in ataxic paraplegia.

In hysterical paraplegia the age and sex of the patient, the peculiar facies, the areas of anæsthesia and hyperæsthesia, and the peculiar gait point to the diagnosis.

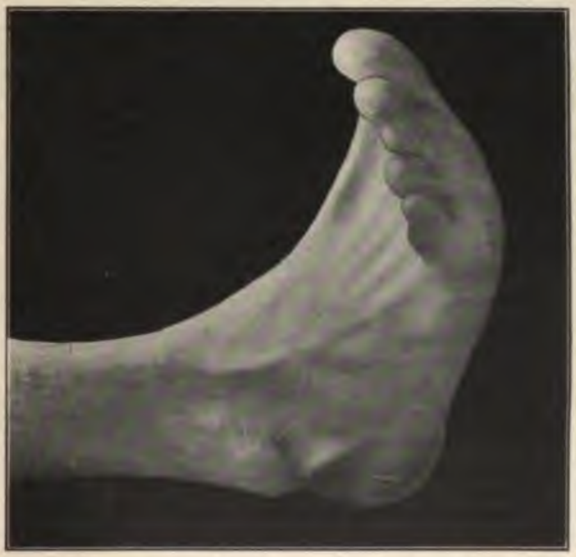
The increased knee-jerk in cases of transverse myelitis occurs when the lesion is situated at such a point in the cord that the lateral tracts are cut off and the reflex arc is preserved.

In neurasthenia the knee-jerks are exaggerated, but are easily exhausted.

Leaving the knee-jerks as a type of a reflex, we find that the skin-reflexes are often lost in cases of apoplexy when the deep reflexes are exaggerated. The table on page 573, from Taylor's *Index of Medicine*, shows the area of skin-reflexes very well.

The plantar reflex obtained by tickling the sole of the foot has recently attained considerable diagnostic importance through the work of Babinski, who has found that in normal persons such irritation causes flexion of the toes on the metatarsus, whereas in disease of the pyramidal tracts extension of the great toe, with or without extension of the other toes, takes place, as, for example, in spastic paraplegia. The normal reflex, somewhat exaggerated, is shown in the figure. It is a noteworthy fact that in infants in health extension takes place instead of flexion, and that the administration of full doses

FIG. 240.



Babinski reflex in health, somewhat exaggerated. The toes are drawn up toward the knee.

of strychnine to adults will sometimes reverse the normal reaction. The Babinski reflex is not pathognomonic of disease of the pyramidal tract, although it is indicative.

In glosso-labio-pharyngeal paralysis the reflexes of the tongue and throat are lost and those of the face sometimes increased; in progressive muscular atrophy the reflexes of the arms are lost, while those of the legs are preserved; and in tubercular meningitis the reflexes are apt to be more marked on one side than on the other.

In athetosis the reflexes are increased in the affected part.

Ankle-clonus is found most marked in lateral sclerosis, in disseminated sclerosis, and in amyotrophic lateral sclerosis. A false clonus is sometimes seen in hysteria.

A TABLE OF THE REFLEXES.

Reflex.	Point of stimulation.	Situation of centre.	Significance.
1. Plantar,	Irritating skin of soles.	Extreme end of cord.	Usual in health.
2. Gluteal,	Irritating skin of buttocks.	Origin of 4th and 5th lumbar nerves.	Rare in health.
3. Cremasteric,	Irritating skin of inner side of thighs.	Origin of 1st and 2d lumbar nerves.	Usual in health; best marked in boys, on account of the newly formed cremaster.
4. Abdominal,	Irritating skin of abdomen in line of nipples.	Origin of 8th to 12th dorsal nerves.	Frequently absent.
5. Epigastric,	Irritating skin of chest in 5th and 6th spaces.	Origin of 4th to 6th dorsal nerves.	May be absent in health.
6. Erector spinæ,	Irritating skin from scapula to crest of ilium.	Origin of all the dorsal nerves.	Rare in health; frequent in wasting disease.
7. Interscapular,	Irritating skin between scapulæ.	Origin of 6th cervical to 3d dorsal.	Rare in health.
8. Palmar,	Palms of hands.	Cervical bulb.	Only in infants.
9. Cranial:			
Conjunctival,	Sclerotic, or inner surface of eyelid.	Medulla.	Absent in disease of 5th nerve only.
Iris (to light),	Pupil.	Anterior portion of oculomotor nucleus.	Absent in disease only.
Palate,	Soft palate and uvula.	Medulla.	Absent in disease only.
Nasal (sneezing),	Naso-respiratory passages.	Medulla.	Absent in disease only.

CHAPTER IX.

SPEECH.

The changes in the speech and voice—Their significance—Aphasia—Apraxia—Alexia—Paraphasia.

THE character of the speech and the tones of the voice often convey a considerable amount of diagnostic information to the physician. While in many diseases no marked alterations from the normal manner of speech are present, in others marked changes take place. Thus, in acute laryngitis due to exposure to cold or irritant vapors the patient has a *whispering* voice. In persons suffering from pulmonary tuberculosis the development of hoarseness and whispering or labored speech tells us only too well of the fact that the grave and distressing complication called laryngeal tuberculosis has arisen, and that the progress of the case will be more rapid toward the fatal result. Again, the sudden onset of whispering voice or complete aphonia, occurring in a young girl whose facies is hysterical, should always arouse a suspicion of hysteria, while if the signs of this condition are absent and the patient has none of the signs of tuberculosis, we should examine the larynx for a papillomatous growth. Again, if *hoarseness* or a whispering voice is manifested by a male of adult years, who is also suffering from dyspnœa, unilateral flushing or sweating of the face and neck, and unequal, rapid radial pulses, we should suspect aortic aneurism or a mediastinal tumor which is pressing on his recurrent laryngeal nerve. There are also other causes of hoarseness due to nervous lesions arising from the long and tortuous course of the nerves supplying it. These have been well grouped together by Felix Semon in the following table. It is interesting to note that in suspected cases of disease of the parts herein named it is well to make a laryngeal examination, since this may reveal a paralyzed cord, although the voice has not indicated such a condition, because by great retraction of the opposite cord the laryngeal opening is kept patulous and phonation is possible.

TABLE SHOWING THE POSSIBLE CAUSES OF LARYNGEAL PARALYSIS.

I. BULBAR AND BULBO-SPINAL AFFECTIONS.	II. PERIPHERAL AFFECTIONS.
1. Hemorrhage and softening.	1. Acute rheumatic influences.
2. Syphilitic processes.	2. Catarrhal neuritis.
3. Tumors.	3. Toxic influences (lead, arsenic, etc.).
4. Diphtheria.	4. Tumors in the posterior cavity of the skull or in the foramen lacerum or foramen jugulare.
5. Progressive bulbar paralysis.	5. Pachymeningitis.
6. That curious form of systemic central nervous disease first described by Hughlings Jackson and Morell Mackenzie, in which one-half of the tongue, the corresponding half of the palate, the corresponding vocal cord, and, in a number of cases, the corresponding trapezius and sterno mastoid muscles are affected.	6. Traumatism (unintentional ligature of nerves, injection of iodine into a goitre, cut throat, stabbing, injury during extirpation of goitre, etc.).
7. Amyotrophic lateral sclerosis.	7. Tumors of neck (goitre, peritracheal glands, etc.).
8. Disseminated cerebro-spinal sclerosis.	8. Aneurisms of the arch of the aorta, innominate, subclavian, carotid.
9. Syringomyelia.	9. Mediastinal tumors (malignant, tuberculous, calcification of bronchial glands, etc.).
10. Tabes dorsalis.	10. Pericarditis.
	11. Pleurisy.
	12. Tuberculosis and pleuritic thickening of apex of right lung.
	13. Chronic pulmonary affections (chronic pneumonia, anthracosis, etc.).
	14. Infectious fevers (typhoid, etc.).
	15. Œsophageal carcinoma.

Hoarseness may also be due to tubercular infection of the larynx, to laryngeal papilloma, and hysteria.

Hysterical mutism may occur in both males and females. It usually follows a fright or violent emotion, or it may follow an hysterical seizure. Sometimes it develops without any such history. The condition lasts from a few hours to months or years, and recovery is often as sudden and unsuspected as the onset. As a rule, the tongue, lips, and jaws are unimpaired in their functions. Sometimes, however, these parts are affected by hysterical spasm. Often there will be hysterical anæsthesia with the mutism. Usually there is no evidence of cerebral lesion in such cases in the sense of impairment of intellect.

When a child speaks with a *nasal twang* or indistinctly we suspect the presence of adenoid vegetations, and will probably find that he or she is addicted to mouth-breathing. Stuttering or stammering may also be due to this cause.

A *feeble, hesitating speech* is often a sign of exhausting disease, and a short and quick but feebly spoken sentence generally indicates that the patient is suffering from some cardiac or pulmonary complaint, which renders him short of breath, so that he hurries through the sentence in order to be able to breathe freely again. In cases of pneumonia or of pulmonary œdema this hurried speech is a very characteristic sign.

Again, in cases of typhoid fever, when the tongue is dry and immobile from accumulated sordes, a mumbling character of the speech is present, even if the brain is entirely clear, and in severe stomatitis the same quality of the voice may be present.

It is in connection with the disorders of the nervous system, however, that the most typical alterations of the voice occur. Let us suppose that a patient in middle life or in more advanced years develops a *slow, scanning speech*, with intention-tremors (see chapter on Hands), nystagmus, and more or less muscular weakness. In all probability he is a sufferer from insular or, as it is otherwise called, disseminated sclerosis. When he speaks each syllable is sharply accentuated and slowly pronounced. The only other condition in which a slow, scanning speech is of great diagnostic importance is in that rare disease, Friedreich's ataxia; but the facts that this disease begins in childhood, that several members of the family are apt to be affected, that there are ataxic symptoms and early talipes equinus, render it easy to separate this affection from insular sclerosis. (See Paraplegia, in chapter on Feet and Legs.)

A *hesitating, halting speech* associated with Argyll-Robertson pupils, unequal pupils, delusions of grandeur, and tremor of the tongue, which last symptom may be so marked as to cause the speech to be indistinct and blurred, is indicative of paretic dementia.

If an incoherent speech develops in a child who is not suffering from an acute illness causing delirium, there will usually be found in association with this symptom the nervous twitchings of chorea, for speech-disturbances occur in about one-third of the patients suffering from this disease.

A very *indistinct speech of a mumbling character*, great difficulty being experienced in the pronouncing of dental and lingual sounds, and perhaps associated with feebleness of the voice, if the larynx is involved, is seen in cases of glosso-labio-pharyngeal paralysis. If the cause of the defective speech be this disease there will be found, as associated symptoms, wasting of the tongue, lingual tremors, some dribbling of saliva from the mouth, and immobility of the lips, the face about the mouth being expressionless.

Somewhat similar symptoms due to paralysis of the lips, with escape of the tongue and pharynx, at least for a long time after labial paralysis develops, are sometimes seen in advanced cases of amyotrophic lateral sclerosis; and a still more close resemblance may be produced by the so-called "pseudo-bulbar paralysis," the lesion of which is in the motor cortex of the brain on both sides, in the lower part of the ascending frontal convolution. Rarely the latter is only a unilateral disease.

A rather *shrill, piping voice*, the sentence being begun with hesitation and then hurried to an end in a rapid volley of words, is sometimes seen in paralysis agitans.

By far the most interesting speech-defect is that called *aphasia.* It is divided into motor aphasia and sensory aphasia.

Before studying these conditions we must discuss the nervous mechanism of speech. When a child learns to talk it performs a purely imitative act. Its auditory nerve conveys the sound to its perceptive centres, and from here an impulse is sent to its motor speech-centres, and these again send impulses to the inferior speech-nuclei in the medulla oblongata, which in turn move the muscles of speech. Simultaneously the child learns the words and stores them in memory-centres for sounds, and also stores in memory-centres "motor memories," which tell him how to repeat the muscular movements a second time. Again, when he learns to recognize objects and call them by name he must use "visual memory" centres. These centres are all best developed in the left hemisphere of the brain in right-handed persons and in the right half of the brain in left-handed persons.

If a person suffers from pure aphasia, he simply loses the memory of how to say certain words, and the lesion is in the third left frontal (Broca's) convolution. This state is sometimes called *aphemia.* The patient knows what a knife is when he sees it, but he cannot recall his motor memories so as to move the muscles to say "knife." He can read to himself, because he has not forgotten the meaning of the words, and for this reason he understands what is said to him, and may be able to repeat a word immediately after it is said, by a purely imitative process. Generally we find with aphasia a condition called *agraphia*, in which the patient cannot write his thoughts, but can copy perfectly. In the great majority of cases of aphasia, however, the patient is paralyzed in his right hand, so that the symptoms of agraphia cannot be demonstrated. Under the name of *paraphasia* we sometimes meet with a condition in which the patient can speak quite freely, but transposes words or interpolates useless words to such an extent that what he says is unintelligible. When the patient slips words, the lesion is in the associating tracts between the speech-centres, and this is called *conduction aphasia.*

In another condition closely connected with aphasia we have a state in which the patient can spell out words from a page set before him, but he cannot read, because the words convey no idea to him. This is called *alexia* or "word-blindness." Again, he may forget the use or significance of certain objects, such as a knife and fork; this is called *apraxia.* Still further, words when spoken to the patient in his native language may be heard perfectly, and yet understood no more than if in some unheard-of language. This is called "word-deafness."

If the patient has simple aphasia, he has a lesion in the third frontal convolution in its posterior part. If he has word-blindness or alexia, the lesion is in the angular gyrus, extending back into the occipital convolution. If he has apraxia or the loss of memory of objects, the lesion is in the same area as in alexia; and if "word-deafness" is present, the lesion is in the posterior part of the first temporal and upper part of the second temporal convolution of the left hemisphere. As the various symptoms of aphasia in all its forms are closely associated with those of focal lesions of the brain, resulting, for example, from hemorrhage or embolism, the reader should read the chapter on Hemiplegia in this connection.

Aphasia is quite frequently met with as a symptom of hysteria, and may occur independently of any organic lesion, so far as we know in children during convalescence from an attack of a severe infectious disease. In the latter cases speech may return many months after.

The following plan of testing a patient, devised by Eskridge from a shorter one by M. Allen Starr, may be followed with advantage:

1. The power to recognize objects seen, heard, felt, tasted, smelt, and their uses.
2. The power to recall the spoken names of objects seen, heard, felt, tasted, and smelt.
3. The power to understand sounds other than speech.
4. The power to understand speech and music.
5. The power to call to mind objects named and point them out at request.
6. If word-deaf, can he recognize his own name when it is spoken?

7. The power to recognize a word spelled aloud.

8. The power to call up mentally the sound of a note, figure, letter, or word.

The examination thus far will test the various sensory areas, but more especially the auditory and the association tracts between the different sensory areas connected with speech.

9. The power to recognize letters, figures, notes, and colors seen.

10. The power to understand printed and written words seen.

11. The power to read printing, writing, and music aloud and inaudibly, and to understand what he reads.

12. The power to recall objects, the names of which are seen.

13. The power to write voluntarily.

14. The power to write at dictation.

15. The power to copy, and the manner of copying, printing, and writing.

16. The power to write the names of objects seen, heard, felt, tasted, and smelt.

17. The power to read aloud and inaudibly, and to understand what has been written.

18. The power to write his name and the ability to read it when written by himself and by another person, or when it is printed.

19. The power to recognize a letter by tracing it with the index-finger or with a pencil, the movements being guided by another.

20. The power to call up mentally the appearance of an object, a figure, a note, letter, or word, when word-blind.

These additional tests will aid in determining the condition of the visual word-memories in the angular gyrus, and the connection between this area and the surrounding sensory and motor areas.

21. The power to speak voluntarily, and, if impaired or lost, the character of the defect.

22. The power to repeat words after another.

23. Does the patient recognize his mistakes in speaking and writing, and can he correct them?

24. Can the patient think in speech (propositionize)?

25. Is there any special difficulty in the use of nouns, verbs, or other parts of speech?

26. The power to understand pantomime or gesture expression.

27. The power to employ intelligently gesture in expression.

28. The power to read figures and to calculate.

29. The power to count both money and in numbers.

30. The power to play a game of cards or other games.

It is not to be forgotten that speechlessness is often present in melancholia and dementia. Further, temporary speechlessness or apparent aphasia may follow severe typhoid fever as a result of cortical exhaustion without the development of hemorrhage, embolus, or thrombus. The prognosis is favorable.

INDEX.

- ABADIE'S sign, 48
- Abdomen, 320
    - causes of distention of, 322
        - of localized bulging of wall of, 327
        - of pain in, 616–623
        - of protrusion of wall, 321
        - of retraction of wall of, 322
        - of swelling in epigastrium, 328
    - frog-belly, 322
    - inequality in shape of, due to meteorism in intussusception, 581
    - inspection of, 321
    - palpation of, 328
    - percussion of, 328
    - "phantom tumor" of, 347
    - regions of, 321
    - scaphoid, 322
    - tumors of, 331
        - position in, 21
- Abscess, cerebral, character of pain in, 612
        - choked disk not generally present in, 497, 578
        - coma in, 554
        - diagnosis of, 578
        - fever in, 578
        - gangrene in, 201
        - symptoms of, 539, 554
        - *tache cérébrale* a sign of, 185
        - vomiting in, 578
    - hepatic, brick-dust sputum in, if communicating with the lung, 602
        - character of fever in, 517, 519
            - of pain in, 620
        - dysentery a cause of, 519
        - hiccough in, 576
        - sweating in, 517, 519
        - symptoms and signs of pyæmic, 352
            - of tropical, 353
        - tongue in, 154
        - vomiting in, 592
    - lumbar, sciatic pain in, 615
    - mastoid, œdema back of the ear in, 209
    - mediastinal, acute, history in, 296
        - cold, 296
        - diagnosis of, 296
        - purulent sputum in, 602
- Abscess, perinephritic, bulging of flank in, 346
    - pulmonary, chronic morning cough in, 597
        - copious and purulent sputum in, 285, 602
        - diagnosis between mediastinal growths and, 296
        - elastic fibres in sputum of, 605
        - following amœbic dysentery, anchovy-sauce sputum in, 602
        - history, signs, and symptoms of, 285
        - rapidity of respiration in, 251
    - pyo-pneumothorax subphrenicus, 342
    - renal, pus-casts in multiple, 420
    - retro-œsophageal. *See* Retro-œsophageal abscess.
    - subphrenic, purulent sputum in, 602
- "Accoucheur's hand" in tetany, 75
- Acetonuria, 439
    - Oppenheimer's modification of Denigé's test for, 439
- Acne due to bromine or iodine, 193
    - due to iron, 194
    - due to working in paraffin, 194
    - of forehead, purulent, in syphilis, 196
- Acromegaly, enlargement of feet in, 128
        - of tongue in, 160
    - face of, 38
    - head in, 53
    - spade-like hand in, 68
- Acroparæsthesia, 236
    - diagnosis of, 237
- Actinomycosis, actinomyces in sputum of, 605
    - as a cause of empyema, 290
    - enlargement of tongue in, 160
- Acuity of vision, changes in, 487
- Acute yellow atrophy of the liver. *See* Yellow atrophy of the liver, acute.
- Addison's disease, appearance of tongue in, 155
    - coma in, 555
    - convulsions in, 570
    - heart in, 318
    - pigmentation of buccal mucous membrane in, 168, 318
        - of skin in, 179, 318
    - symptoms of, 179, 593

Addison's disease, vomiting in, 593
Adherent pericardium, symptoms of, 255
Adhesive pericarditis, symptoms of, 351
Adiposis dolorosa, 210
Ægophony over pleural effusions, 279, 288
Æsthesiometer, 212
Agraphia, 634
  location of lesion in, 635
Ainhum, 135
Air-passages, foreign bodies in, 294
"Alar chest" of phthisis, 245
Albuminometer, 433
Albuminuria, anæmic, 434
  boiling test for, 430
  cyclic, 434
  due to renal congestion, 434
  Esbach's test for, 433
  Heller's test for, 431
  in acute diffuse nephritis, 445
  indicative of renal disease, 434
  marked, or absent, in amyloid kidney, 447
  potassium ferrocyanide test for, 432
  quantitative test for, by centrifuge, 432
  transient, in chronic interstitial nephritis, 447
  trichlor-acetic acid test for, 432
Albuminuric retinitis, 499
Albumosuria, 434
  in Bradshawe's disease, 434
  in normal puerperium, 434
  myelopathic, 434
  said to be due to the action of micro-organisms, 434
Alcoholism, convulsions due to, 561, 564
  facial expression in, 28
  hyperæsthesia in chronic, 239
  morning vomiting in, 588
  symptoms of acute, 553
Alcohol-poisoning, tremors in, 80
Alexia, 635
  location of lesion of, 635
Alkaptonuria, 439
Allochiria in hysteria, 236
  in multiple sclerosis, 236
  in myelitis, 236
  in paralysis agitans, 236
  in tabes dorsalis, 236
Amaurosis, 500
  significance of, in brain fever, 537
  uræmic, 500, 577
Amaurotic family idiocy, 104
Amblyopia, 497
Ameba coli, 455, 456
Amebic dysentery, 455
Amphoric breathing, rarely heard in pneumothorax, 274
    in small cavity, 274
Amyloid disease of kidney, 447
Amyloid disease of kidney, hyaline, fatty, and granular casts in urine in, 447
    syphilis, prolonged suppuration, and extensive bone disease accompanying, 447
    urine in, 447
  of liver, enlargement and smoothness of liver in, 349
    history of prolonged suppuration elsewhere in, 351
    jaundice occurring in, 173, 174
Amyotrophic lateral sclerosis, changes in shape of hands in, 73
Anæmia, 383
  albuminuria in, 434
  appearance of nails in, 59
    of tongue in, 155
  bothriocephalus latus a cause of, 462
  cerebral, choked disk in, 578
    vertigo in, 547
    vomiting in profound, 578
  dropsy of feet and legs in, 207
  headache in, 546
  hiccough in, 576
  hyperæsthesia in, 239
  in acute diffuse nephritis, 446
  in anchylostomum duodenale, 385, 463
  in atrophy of gastric tubules, 384
  in bleeding hemorrhoids, 384
  in cancer, 385
  in chronic indigestion, 384
    lead- or arsenic-poisoning, 385
  in convalescence from infectious disease, 383
  in gastric ulcer, 385
  in hemorrhage, 384
  in malaria, 385
  in pyelitis, 448
  in renal disease, 385
  in sarcoma, 385
  in tapeworm, 385
  in tuberculosis, 385
  in uræmic poisoning, 385
  œdema in, 207, 208
  pallor of conjunctiva in, 464
  papillitis in, 497
  pernicious, cardiac palpitation in, 384
    change in red blood-corpuscles in, 384
    chills followed by fever in, 519
    fatty degeneration of heart in, 318
    gradually increasing dyspnœa in, 384
    indicanuria in, 415

Anæmia, pernicious, jaundice in, 175
    marked pallor without loss of flesh in, 384
    pulsating cervical vessels in, 253
    venous murmurs in, 384
    vertigo in, 384
  purpuric eruptions in, 184
  splenic, 356
    blood changes in, 356
Anæmic heart-murmur, 300
  necrosis, 318
Anæsthesia, 216
  bilateral, in Friedreich's ataxia, 222
    in hemorrhage of the cord, 223
    in hysteria, 221
    in injuries of the cord, 221
    in lesions of the pons, 221
    in locomotor ataxia, 221
    in meningitis, spinal, 221
    in myelitis, 222
    in spinal disease, 221
    in syringomyelia, 223
    never in cerebral lesions, 221
  causes of, 216
  crossed, in lesions of one side of the cord, 220
    of peduncle, 219
    of pons, 220
  differential diagnosis in facial, 235
  distribution of, in special nerve involvement, 232
  dolorosa in cancer of spine, 223
  gauntlet or stocking form of, in hysteria, 219
  in 50 per cent. of neuritis due to diphtheria, 231
  in hysteria, 216
  location of lesion of, in the spinal cord, 224, 225
  neuritis as cause of, 228
  of body and legs in locomotor ataxia, 221
  of face, 235
  of Friedreich's ataxia, 222
  of myelitis, 222
  of toxic peripheral neuritis, 230
  patches of, in syringomyelia, 223
  reflexes preserved in cerebral, 219
  signs of, due to neuritis, 229
  unilateral, 218
  varieties of, 216
  zones of, 223
Analgesia in locomotor ataxia, 221
  in Morvan's disease, 72
Anasarca, general. *See* General anasarca.
Anatomy of brain, 136
  of optic nerve, 489
  of sensory tracts, 215, 223
  of tongue, 150
Anchovy-sauce sputum, 602
Anchylostomum duodenale, anæmia due to, 385, 463
Aneurism, aortic, blood-streaked sputum due to leakage from aorta in, 601
    bruit in, 305
    bulging of chest in, 246, 307
    hæmoptysis in, 601
    pain in, 307, 601, 614
  appearance of hands in, 58
  brassy cough due to pressure on larynx by, 307, 598
  bruit in, 305, 307
  dyspnœa or dysphagia in, 164, 307
  enlarged cervical vessels in, 253
  hemorrhagic pleural effusion in, 291
  history of syphilis in, 306
  hoarseness of voice due to pressure on recurrent laryngeal nerve by, 306, 631
  intracranial, headache due to, 546
  laryngeal spasm due to pressure on recurrent laryngeal nerve by, 294, 306
  loss of pulsation in peripheral vessels in, 309
  œdema of upper extremities in, 209
  of abdominal aorta, 343
  pain in abdominal, 621
  pressure on bile-duct causing jaundice in, 173
  sweating of side of head in, 205
  symptoms of, 305
  thoracic, diagnosis of, from mediastinal growths, 295
    pain in, 296
  tracheal tugging in, 308
Angina pectoris, causes of, 361
    character and position of pain in, 613
    symptoms of, 613
Angioneurotic œdema, appearance of eyelids in, 209
      of hands in, 67
      of skin in, 211
Ankle-clonus, how best elicited, 626
  in amyotrophic lateral sclerosis, 630
  in ataxic paraplegia, 98
  in hysteria, 630
  in lateral sclerosis, 630
  most marked in disseminated sclerosis, 630
Ankylostomum duodenale, 462
Annulus migrans, 159
  senilis, 465
Anthrax maligna, anthrax bacilli found as cause of, 527
    character of fever in, 527
    symptoms of, 199, 527
  simplex. *See* Carbuncle.
Antidiphtheritic serum, roseola due to injection of, 190

Anus, fissure of, diarrhœa in, 453
Aorta, aneurism of abdominal, 343
    of descending, symptoms of, 307
    of greater curvature of ascending, symptoms of, 307
    of transverse portion of arch of, symptoms of, 307
Aortic aneurism, bruit in, 305
    diagnosis of, by fluoroscope, 309
    pulse in, 305
  regurgitation, 304
    “ox-heart” of, 305
    pulse in, 363
    Quincke's sign of, 313
    “water-hammer” pulse in, 305
  stenosis, 304
    pulse in, 363
    reduplication of heart-sounds in, 299
    symptoms of, 312
  valvular disease, 304
Aortitis, acute, causes of, 613
    character of pain in, 613
Apex-beat, 256
Aphasia, 634
  due to hæmatoma near island of Reil, 146
  due to hemorrhage into island of Reil, 146
Aphemia, 634
Aphonia due to pressure in aortic aneurism, 306
  in hysteria, 631, 632
Aphthongia, spasm of tongue in, 162
Apoplexy, cause of, 361
  Cheyne-Stokes breathing in, 252, 552
  coma in, 552
  conjugate lateral paralysis of ocular muscles in, 480
  contractions following, 74
  diagnosis between coma of, and alcoholism, 553
  epilepsy associated with, 558
  ingravescent, 144
  involuntary passage of urine in, 406
  knee-jerk exaggerated some time after shock, 628
      decreased immediately after shock, 627
  paralytic chorea following stroke of, 77
  peptonuria in, 435
  skin-reflexes lost when deep reflexes are exaggerated, 629
  vertigo before, 547
Appendicitis, character of fever in, 528
    of vomiting in, 585
  leucocytosis in, 389
  pain in, 617
  symptoms of, 330
Appetite, wayward, in chlorosis, 385
Apraxia, 635
  location of lesion in, 635
Arcus senilis, 318, 465
Argyll-Robertson pupil, 92, 484
    not present in ataxic paraplegia, 98
        in Friedreich's ataxia, 95
    present in diabetes mellitus rarely, 92, 484
        in true ataxia, 484
Argyria, 178, 179
Arhythmia in cardiac dilatation, 314
Arms, movements of, 73
  spastic rigidity of, an early sign of
    tremors of, 79
Arrested development, 103
    cerebral spastic paraplegia in, 103
Arsenical neuritis, 80
Arterial disease, ophthalmoscopic evidence of, 499
  sclerosis, general, accentuation of heart-sounds in, 298
  tension, 361
    causes of high, 361
      of low, 363
    high in acute diffuse nephritis, 446
Arteritis, symptoms of syphilitic, 538
Arthralgic form of gonorrhœal arthritis, 130
Arthritis deformans, distortion of fingers in, 61, 64
    enlargement of joints in, 131
    formation of osteophytes in, 131
    Heberden's nodes in, 65
    knee-jerk increased in, 628
    seal-fin hand of, 65
  gonorrhœal, 129
  multiple, in dengue, 133
  of cerebrospinal meningitis, 132
  septic, appearance of hands in, 67
    changes of joints in, 132
Arthropathies, 130
Ascarides, enlargement of gall-bladder due to, 353
Ascaris lumbricoides, 461, 462
Ascites, apex-beat raised in, 257, 324
  caput Medusæ in hepatic cirrhosis causing, 327
  causes of, 324
  diagnosis of, 324
  examination of withdrawn fluid in, 325
  rapid respiration in, 253
  skin of abdomen in, 203
Astasia abasia, 101
Astereognosis, 213
Asthenic bulbar paralysis, 47. *See* Paralysis, asthenic bulbar.

Asthma, catarrhal pneumonia following, 294
character of râles in, 293
due to cardiac lesions, 294
due to gastric disorders, 294
due to reflex nasal irritation, 294
due to renal disease, 294
erect position in, 293
expiration prolonged in, 252, 293
physical signs and symptoms of, 293
rapid respiration in, 252
sputum of, Charcot-Leyden crystals in 294, 603
Curschmann's spirals in, 294
pearls of mucus in, 603
Asymmetry, facial, 35
Asynchronism of the ventricles, reduplication of first sound of heart due to, 299
Ataxia, differential diagnosis of, 96
Friedreich's. *See* Friedreich's ataxia.
hereditary cerebellar, 96
spinal. *See* Friedreich's ataxia.
locomotor. *See* Locomotor ataxia.
Ataxic paraplegia, 98
Atheroma, headache in, 535
in chronic interstitial nephritis, 447
in old age, 361
in syphilis, 361
symptoms of, 361
vertigo in, 547
Athetosis, 82
reflexes increased in, 630
Atrophy, acute yellow, of the liver, coma in, 551
jaundice in, 173, 175
vomiting in, 593
bilateral, of tongue, causes of, 159
facio-humero-scapular type of, 32
idiopathic muscular, ptosis in, 44
of gastric tubules, anæmia in, 384
of nails, 59
optic, 497
progressive muscular, 69
Aran-Duchenne type of, 129
claw-hand in, 69
condition of reflexes in, 630
excessive sweating of hand in, 60
fibrillary twitch in, 82
leg-symptoms of, 108, 129
-type of, 129
peroneal leg-type of, 129
unilateral, of tongue, in chronic lead-poisoning, 159
Aura, 556
Auscultation of heart and vessels, 297
of respiratory apparatus, 271
Auto-intoxication, headache in, 533
poisons causing, 533
symptoms of, 533

BABINSKI'S reflex, 629
Baccelli's sign, 278
Bacilli, anthrax, 527
in preputial smegma, 428
in stools in cholera, 452
in tubercular pyelitis, 448
of Eberth, in stools of typhoid fever, 509
tubercle, in sputum, methods of examining for, 606–608
in urine, 428
Bacillus typhosus as a cause of empyema, 290
Banti's disease, 357
symptoms of, 357
Barrel-shaped chest in emphysema, 246, 292
Basedow's disease, tremor of hand in, 81
Bedsores in acute transverse myelitis, 202
in hemiplegia, 202
Behavior in advanced hepatic and renal cirrhosis, 20
in opium-poisoning, 21
of child with cerebral trouble, 24
of healthy child, 24
Bergeron's chorea, 78
Beri-beri, general anasarca in, 207
Biceps tendon reflex, how developed, 626
Bilateral anæsthesia, 221
facial paralysis, 46
ptosis, 45
Bile-ducts, obstruction of, causes of, 353, 354
Bile in urine, test for, 440
Bilious remittent fever. *See* Malaria, remittent.
Biliousness, appearance of tongue in, 152
Bismuth, odor of breath after administration of, 19
Black vomit in yellow fever, 521
Bladder, causes of incontinence of urine in the, 405
of retention of urine in the, 402
character of blood in urine from the, 411
nervous, 406
paralysis of, 403
stone in, 406
symptoms of disease of, 402
of inflammation of, 405
of locomotor ataxia, 404
-symptoms of myelitis, 403
of paretic dementia, 404
of tabes, 404
tumors of, obstructing urinary flow, 406
Blepharo-facial spasm, 48
Blindness, causes of, 486, 487
sometimes from influence of drugs, 500
Blood casts in urine, 420
changes in, in infectious diseases, 389

- Blood, circulation of, 360
  - diagnostic value of, in appendicitis, 389
  - estimation of hæmoglobin in, by hæmoglobinometer of v. Fleischl, 376
  - in sputum, 601
  - in stools, 457, 459, 584
    - character and significance of, 452
    - in dysentery, 455, 456
    - in entero-colitis, 454
  - in urine, 409
    - guaiacum test for, 413
    - Heller's test for, 413
    - in severe renal disease, 621
  - in vomitus, 586, 587
  - method of examining, 369
    - of staining with Ehrlich's triple stain, 382
  - normal constitution of, 367
  - parasites of, 389
  - red corpuscles of, changes in, in anæmia, 383
    - coloring-matter of, decreased in oligochromæmia, 368
    - decreased in myelogenous leukæmia, 387
      - in oligocythæmia, 367
    - estimation of, by hæmatocytometer, 370
      - with hæmatocrit, 375
    - extraordinary decrease of, in pernicious anæmia, 384
    - increased in polycythæmia, 367
    - megaloblasts, 384
    - megalocytes, 368
    - microcytes, 368
    - normoblasts, 368
    - nucleated, 368
    - poikilocytes, 368
    - proportion of, to white, 368
    - shadow-corpuscles, 368
    - varieties of, 367
  - -streams of the retina, influence of arterial pressure on, 499
  - sugar in, 400
  - -vessels and pulse, 359
  - white corpuscles of, amœboid movements of, 381
    - eosinophiles, 382
    - estimation of, with hæmatocytometer, 373
    - increase of, 381, 386
    - lymphocytes, 381
      - alone increased in lymphatic leukæmia, 388
    - mononuclear leucocytes, 381
    - multinuclear leucocytes, 382
    - neutrophiles, 382
- Blood, white corpuscles of, polymorphous leucocytes, 381
    - polynuclear leucocytes, 381
    - proportion of, to red, 368
    - Toison's solution for counting, 373
- Blowing breathing, 273. *See* Bronchial breathing.
- Blue line on gums in lead-poisoning, 163
- Boiling test for albumin in urine, 430
- Boils in diabetes mellitus, 199
  - in paraffin-workers, 199
  - in tar-workers, 199
- Bothriocephalus latus, 462
- Bowels, cancer of, 343
  - consideration of, 449
  - constipated, in jaundice, 172
  - patient's idea as to regularity of, 23
  - sloughs of, in intussusception, 460
- Brachial monoplegia, 84
  - apparent, 88
  - bilateral, 88
  - causes of cortical lesions in, 85
  - due to cortical lesions, 84
  - due to crutch-paralysis, 88
  - due to fracture or dislocation of the head of the humerus, 87
  - due to growth in neck or axilla, 87
  - due to hysteria, 86
  - due to injury of the brachial plexus or important branches, 86
  - due to lead-poisoning, 88
  - due to locomotor ataxia, 87
  - due to primary brachial neuritis, 86
  - Erb's paralysis, 87
  - Klumpke's paralysis, 88
  - paralysis of special muscles in, 86, 88
  - signal-symptoms of cortical, causes of, 85
  - paræsthesia, 89
- Bradshawe's disease, albumosuria in, 434
- Bradycardia, 317, 366
  - in jaundice, 317
  - in typhoid fever, 317
- Brain, areas of, 136
  - tumors of, anæsthesia in, 218
    - Cheyne-Stokes breathing in, 252
    - convulsions in, 536
    - diagnosis of, 536
    - differential diagnosis between chronic nephritis and, 537
    - headache in, 536
    - hyperæsthesia in, 238
    - papillitis of optic nerve in, 496, 536
    - paræsthesia in, 236
    - paralysis in, 536
    - slow breathing in, 537

Brain, tumors of, slow pulse in, 537
  vomiting in, 536
Bramwell's method of staining and mounting casts in urine, 424
Breakbone fever. *See* Dengue.
Breast, pigeon-, 247
Breath after the administration of bismuth, 19
  in bronchiectasis, 19, 286
  in chronic atrophic nasal catarrh, 19
  in diabetes mellitus, 19
  in diphtheria, 19
  in empyema opening into bronchus, 19
  in fever, 19
  in gangrenous stomatitis, 19
  in gastric disorders, 19
  in ozæna, 19
  in pulmonary gangrene, 19, 286
  in tonsillitis, 19
  in uræmia, 19
  shortness of, in heart disease, 312
Breathing, abdominal, 253
  amphoric, 274
  blowing, 273
  bronchial, 271, 273
  broncho-vesicular, 272
  cavernous, 274
  cog-wheel, 273
  costal, 253
  puerile, 272
  tubular, 271, 273
  vesicular, 271, 273
Bright's disease. *See* also Kidneys and Nephritis.
    complications of, 446
    obstinate cough in, 598
    retinal hemorrhage in, 500
    retinitis in, 500
    roseola in, 189, 191
    secondary diphtheritic dysentery in, 456
    sputum in, 602
    value of estimation of urea in, 444
Broadbent's sign of adherent pericardium, 255
Bromidrosis, cold, clammy hands in, 60
  in hysteria, 205
Bronchial breathing, 271, 273
    due to compression of lungs over pleural effusion, 273
    in catarrhal pneumonia, 273
    in cavity, 273
    in consolidation, 273
Bronchiectasis, fetid breath of, 19, 286
  purulent sputum in, 602
  sacculated, loose morning cough in, 597
Bronchitis, acute, character of sputum in later stages of, 600
  bronchorrhœa in, 292
Bronchitis, bubbling râles in, 275
  Charcot-Leyden crystals in, 603
  chronic, in chronic interstitial nephritis, 447
  Dittrich's plugs in sputum of putrid, 292
  fever in, 526
  fibrinous, small casts of bronchioles coughed up in, 603
  hard, dry cough in, 291
  moderate, early in typhoid fever, 509
  physical signs of, 292
  purulent, crystals of margaric acid in sputum of, 605
  putrid, 292
  râles in, 292
  rapidity of respiration in severe, 251
Bronchophony in pleural effusions, 288
  in tuberculosis of the lungs, 284
Broncho-vesicular breathing, 272
Brow ague in malaria, 546, 611
"Bruit" in aortic aneurism, 305
Bullæ, due to antipyrin or iodine, 200
  due to pemphigus, 200
  due to trophic lesions in disease of central nervous system, 200
Burns, hæmoglobinuria after severe, 413
Bursal and synovial form of gonorrhœal arthritis, 130

CACHEXIA, general anasarca in cancerous, 207
Calculus. *See* also Stone.
  pancreatic, pain in, 620
  renal, character of pain in, 621
    vomiting in, 593, 621
  salivary, 165
  vesical, symptoms of, 406, 621
Caput coli, diseases of the, 358
  Medusæ, 327
Carbol-fuchsin stain for tubercle bacilli, 607
Carbuncle, 199
Carcinoma, appearance of skin in, 180
  cheesy pallor of face in, 178
  expression of face in, 29
  gastric, anæmia in, 385
    character of vomiting in, 586
    coffee-ground vomit in, 332, 586
    fever in, 519
    indicanuria in, 415
    most frequent at pylorus, 332
  hemorrhagic effusion in pleura in, 290
  melanotic, black urine in, 409
  œsophageal, brassy cough due to pressure on larynx by, 598
  of bowel, 343
  of caput coli, 358
  of gall-bladder, 355
  of liver, indicanuria in, 415
  of pancreas, diabetes mellitus in, 334

- Carcinoma of pancreas, jaundice in, 173, 334
    - oily stools in, 334
    - symptoms of, 334
  - of pylorus, diagnosis of, 331
      - between gastric ulcer and, 333
    - dilatation of stomach due to, 332
    - excess of lactic acid in stomach in, 338
    - symptoms of, 332
  - renal, hæmaturia in, 410
  - retraction of abdominal wall in, 322
  - visceral, peptonuria in, 435
- Cardiac crises, 318
  - dulness, alterations in, due to valvular lesion, 270
    - increase of, due to hypertrophy or dilatation of heart, 266
      - due to pericardial effusion, 267
    - lessened by emphysema, 269
    - normal extent of, 265
  - feebleness, 318
  - triangles, 265
- Cardio-pulmonary murmur, 299
    - diagnosis of, 300
- Caries of bones of skull, headache in, 546
  - of cervical vertebræ, retraction of head in, 51
  - of teeth in pregnancy and diabetes mellitus, 163
  - of vertebræ, paraplegia due to, 103
  - spinal, signs or symptoms of, 346, 615
- Carphologia, 60, 81
- Carpo-pedal spasm in rickety, hydrocephaloid children, 75
- Casts, Bramwell's method of mounting and staining, 424
  - centrifuge in examining, 419
  - composition of, 418
  - Farrant's solution for staining, 424
  - fatty, 422
  - hyaline, 422
  - of blood-cells, 420
  - of bronchioles found in sputum, 603
  - of epithelial cells, 419
  - of granular matter, 421
  - of micrococci, 421
  - of pus-cells, 420
  - resemblance of cylindroids to, 422
- Catalepsy, 32
  - expression in, 32
- Cataract, congenital, nystagmus in, 482
  - sometimes the result of diabetes mellitus, 499
- Catarrh, acute gastric, appearance of tongue in, 153, 588
      - symptoms of, 588
      - vomiting in, 588
    - rectal, symptoms of, 454, 455
- Catarrh, chronic gastric, morning vomiting of drunkards in, 588
      - vomiting due to excessive tea-drinking in, 588
  - gastro-intestinal, fever in mild, in children, 505
    - frog-belly in children with, 322
    - uric acid excess in urine of, 425
  - laryngeal, spasm due to, 294
  - nasal, character of headache due to, 546
  - of middle ear in syphilis, 163
  - persistence of fever in measles due to bronchial or gastro-intestinal, 514
  - respiratory cough at night in, 599
- Catarrhal fever. *See* Influenza.
  - pneumonia. *See* Pneumonia, catarrhal.
- Cavernous breathing, 274
  - sinus, thrombosis of, 543
- Cavity of lung, bronchial breathing in, 273
    - Hippocratic succussion in large, partly filled with liquid, 279
    - pectoriloquy in, 279
    - tubercular, purulent sputum in, 602
- Centrifuge, 374
  - in examination of blood, 375
    - of urine, 419
  - quantitative test for albumin with, 432
- Cephalalgia. *See* Headache.
- Cerebellar ataxia, hereditary, 96
  - hemorrhage, 146
  - titubation, 101
- Cerebral abscess, coma in, 554
    - pain in, 612
    - papillitis in, 497
  - effusion, 53
  - embolism, brachial monoplegia in, 85
  - palsy of children, 73
      - athetosis in, 82
      - epileptiform convulsions in, 74
      - flexion of hands in, 73
      - gait in, 99
      - intention-tremor in, 74
      - knee-jerk in, 628
  - softening, coma of, 553
    - symptoms of, 553
  - spastic paraplegia, 103
  - syphilis, coma in, 554
  - thrombosis, œdema in, 209
  - tumor, pain in, 612
- Cerebro-spinal fever, Cheyne-Stokes respiration in, 252
  - meningitis, 522
    - character of fever in, 522
    - convulsions in, 522

- Cerebro-spinal meningitis, diagnosis between croupous pneumonia, otitis media, tubercular infection, and, 523
    - epidemic, coma in, 554
    - headache in, 522
    - herpes labialis in, 199
    - hyperæsthesia in, 238
    - joints in, 132
    - lumbar puncture in diagnosis, 522
    - peptonuria of great importance in diagnosis of, 435
    - petechiæ in, 184
    - rigidity of back of neck in, 522
    - vomiting in, 522
  - sclerosis, multiple, 106
- Cestodes, 460
- Chancre of tongue, 156
- Changes in acuity of vision, 487
- Charcot-Leyden crystals in acute bronchitis, 603
    - in asthmatic attacks, 294, 603
    - in chronic croupous pneumonia, 603
    - in pulmonary tuberculosis, 603
- Charcot's fever, 175, 517
- Cheek, swelling of, in noma, 165
    - in peliosis rheumatica, 165
    - in salivary calculus, 165
    - in Schönlein's disease, 165
    - in stomatitis, 165
- Chenzynski's solution for staining malarial organisms, 396
- Chest, auscultation of, 271
  - barrel-shaped, of emphysema, 246
  - beaded ribs of, in rickets, 249
  - bulging of, causes of, 246
    - of sternum in pigeon-breast, 247
  - expansion, 253
  - friction sounds in, 276
  - Harrison's grooves in, 247
  - inspection of abnormal, 245
    - of normal, 241
  - localized bulging of, causes of, 246, 253
  - pain in, shown by pinched nose in children, 30
  - palpation of, 255
  - percussion of, 258
  - phthisical or alar, 245
  - resonance of, 259
  - shrinking of, 249
  - variation in shape of, in health, 244
  - -wall, pulsations on, causes of, 254
- Chewing or sucking movements in meningitis, 541
- Cheyne-Stokes respiration, causes of, 252
    - prognostic value of, 252
    - pupillary reaction associated with, 487
- Chickenpox, appearance of eruption, in 197
  - character of fever in, 515
  - disparity between height of fever and degree of illness in, 515
- Children, ages at which the different teeth appear. 163
  - appearance of hands of, in heart disease, 58
  - causes of fever in, 505
  - cerebral palsy of, 72
  - congenital malformation of heart in, 319
  - coryza in, 24
  - crying of, 24
  - earache in, 24
  - empyema in, 289
  - frog-belly in, 322
  - gastro-pulmonary fever of, 530
  - grinding of teeth by, significance of, 164
  - hydrocephaloid disease of, 570
  - incontinence of urine in, 405
  - languor in cerebral trouble of, 24
  - nystagmus in, 482
  - pleurisy in, 24
  - pneumonia in, 24
  - poliomyelitis in, 122
  - respiration of, 24
  - rose-rash in, 187
  - stomatitis in, 24
  - syphilis in, 24
  - tubercular meningitis in, 541
  - typhoid fever in, 520
  - winning confidence of, 23
- Chill in acute pleurisy, 286
  - in croupous pneumonia, 525
  - in intermittent malarial fever, 515
  - in pernicious anæmia, 519
  - in pyelitis, 448
  - in septic poisoning, 519
  - slight, in dysentery, 455
- Chills, 501
- Chilly sensations and nausea in acute diffuse nephritis, 445
- Chin-jerk, how elicited, 626
- Chin, position of, in wry-neck, 51
- Chlorosis, constipation in, 385
  - disorders of menstruation in, 385
  - dyspnœa in, 385
  - greenish-yellow color in, 178
  - hæmoglobin decreased in, 385
  - humming-top murmur in jugular vein in, 385
  - irregular heart in, 385
  - peculiar pallor of, 28, 180
  - poikilocytosis in, 385
  - slight fever in, 385
  - wayward appetite in, 385
- Choked disk, 496
    - in cerebellar tumor, 496, 578
    - in cerebral tumor, 496, 578

- Choked disk in profound cerebral anæmia, 578
  - not generally seen in cerebral abscess, 578
- Cholangitis, catarrhal or suppurative, character of fever in, 519
  - gallstone colic in, 519
  - leucocytosis in, 519
  - marked jaundice in, 519
- Cholecystitis, acute, pain in, 619
- Cholelithiasis, 354
- Cholera Asiatica, comma bacillus in, 452
    - diagnosis of, 452
    - dryness of skin in, 205
    - Hippocratic face in impending death in, 35
    - indicanuria in, 415
    - roseola in, 189, 191
    - subnormal temperature in, 530
    - systemic infection in, 452
    - temperature in, 527
    - vomiting in, 585
  - infantum, character of fever in, 527
    - Cheyne-Stokes respiration in, 452
    - cold skin and high rectal temperature in, 527
    - diarrhœa in, 527
    - disparity between axillary and rectal temperatures in, 452
    - mousy, musty odor of stools in, 458
    - obstinate vomiting in, 586
    - sinking in of fontanelles in, 54
    - subnormal temperature in severe, 530
    - symptoms of, 451
  - morbus, subnormal temperature in severe, 530
    - symptoms of, 451, 452
  - nostras. *See* Cholera morbus.
- Chorea, anæmic murmurs in children in, 300
  - Bergeron's, 78
  - character of spasms in, 572
  - diagnosis of, 572
  - electric, 78, 573
  - expression in, 32
  - facial spasm in, 48
  - habit-, 77
  - heart disease in, 300, 301
  - hemianæsthesia in, 217
  - hereditary, 78
  - Huntington's, 78, 573
  - hysterical, 78
  - incoherent speech in, 633
  - insaniens, 573
  - loss of knee-jerk in, 627
  - major, 573
  - maniacal, 77
  - minor, movements in, 77
  - nodding spasm in, 50
- Chorea, paralytic, 76, 77
  - post-hemiplegic, lesion of, 147
  - senile, 77
  - spasm of face in, 48
    - of tongue in, 162
- Chromogen in urine, test for, 415
- Chronic hydrarthrosis, 130
  - lead-poisoning, paralysis in, 76
  - poliomyelitis, claw-band in, 72
  - superficial glossitis, 158
- Chvostek's symptom of tetany, 571
- Chyluria, caused by filaria sanguinis hominis, 398, 415
  - test for, 415
- Circulation of the blood, 360
- Cirrhosis, hepatic, ascites in, 207, 324, 351
    - caput Medusæ in ascites caused by, 327
    - disorders of digestion in, 351
    - enlargement of spleen in, 356
    - hæmatemesis in, 351, 587
    - jaundice in, 173
    - symptoms due to, 351
    - uric acid in excess in urine of, 425
- Claw-foot, 108
  - in Freidreich's ataxia, 109
  - -hand, 69
- Clothing, diagnostic significance of, 17, 18
- Clubbed fingers, 58, 312
- Coffee-ground vomit in gastric cancer, 586
    - in locomotor ataxia, 586
    - in phosphorus-poisoning, 591
- Cog-wheel breathing, 273
- Coin percussion in pleural effusion with pneumothorax, 288
- Cold abscess of mediastinum, 296
- Colic, causes of abdominal, 616
  - character of crying in children caused by abdominal, 24
  - gallstone, in catarrhal cholangitis, 354, 519
  - hepatic, 172, 619
  - lead, pain in, 620
  - renal, character of pain in, 621
    - diagnosis of, 621
  - retraction of abdominal wall in renal, hepatic, and lead, 322
  - sometimes due to strongylus gigas, 412
  - ureteral, 409
- Collapse, profuse sweating a sign of, 204
- "Collar of brawn" in scarlet fever, 513
- Colon bacillus as a cause of empyema, 290
- Coma, ability to protrude tongue in, when other orders fail to gain response, 160
  - causes of, 548
  - following pernicious malarial fever, 552
      - position in, 20

- Coma in acute alcoholic poisoning, 548
  - differential diagnosis of, 549
  - yellow atrophy of liver, 551
  - in Addison's disease, 555
  - in apoplexy, 552
  - in cannabis-indica-poisoning, 549
  - in cerebral abscess, 554
    - disease, 20
    - softening, 553
    - syphilis, 554
  - in chloral-poisoning, 550
  - in chronic parenchymatous nephritis, 446
  - in cirrhosis of liver, 351
  - in diabetes, 447, 551
  - in epidemic cerebro-spinal meningitis, 554
  - in general paralysis, 555
  - in heart-failure, 555
  - in heat-stroke, 555
  - in multiple sclerosis, 555
  - in opium-poisoning, 549
  - in *petit mal*, 555
  - in purulent leptomeningitis, 554
  - in Raynaud's disease, 554
  - in renal disease, 20
  - in subdural hemorrhage, 553
  - in thrombosis of brain-sinuses, 553
  - in typhoid fever, 551
  - in uræmia, 550
  - rapidity of respiration in diabetic or uræmic, 252, 551
  - slowness of breathing in uræmic or diabetic, 252, 551
  - -vigil, 551
- Concomitant squint, 467
- Congenital cataract, nystagmus in, 482
  - lymphangioma, tongue in, 159
  - malformations of heart, 319
    - rules for diagnosis of, 319
- Congestion, hepatic, tenderness in, 351
  - pulmonary, 286
    - physical signs and history of, 286
  - renal, albuminuria in, 434
    - blood-casts in, 411, 420
    - irritation of bladder in, 405
- Consciousness, loss of, in cerebral hemorrhage, 144
- Constipation, causes of, 449
  - in chlorosis, 385
  - in chronic lead-poisoning, 450
  - in diabetes insipidus, 449
    - mellitus, 449
  - in intestinal obstruction, 450, 579, 582
  - in jaundice, 449
  - in phosphorus-poisoning, 449
  - in reflex irritation, 450
  - indicanuria in, 415
- Continued fever. *See* Malaria, remittent.
- Contraction of pupil of the eye, 465
- Contractures in hysteria, 74, 111
- Convulsions or general spasms, 556
  - in acute articular rheumatism with hyperpyrexia, 528
  - in Addison's disease, later stages, 570
  - in anterior poliomyelitis, 529
  - in brain-tumor, 536
  - in cerebro-spinal meningitis, 522
  - in uræmia, 577
  - clonic, in acute alcoholism, 561
    - in chronic lead-poisoning, 561
    - in epilepsy, 556
    - in eruptive fevers, 561
    - in general paresis, 95
    - in hysteria, 565
    - in irrigation of pleural cavity, 570
    - in Jacksonian epilepsy, 557
    - in malaria, 563
    - in malingerers, 569
    - in multiple sclerosis, 570
    - in post-hemiplegic epilepsy, 558
    - in puerperal eclampsia, 568
    - in syphilitic epilepsy, 560
    - in syncope, 569
  - epileptiform, in cerebral palsy of children, 74
  - erotic, in phosphorus-poisoning, 592
  - of infants in meningitis, 570
    - in pseudo-meningitis, 570
    - in reflex irritation, 569
  - salaam, 573
  - tetanic, in acute yellow atrophy of liver, 572
    - in hæmatoma of dura mater, 572
    - in strychnine-poisoning, 570
    - in tetanus, 570
    - in tetany, 571
- Convulsive tic, 78
  - facial spasm in, 48
- Coprolalia, 49, 78
- Coproliths, 460
- Corneal ulceration in scrofulosis, 499
- Corpuscles of Ehrlich, 384
- Corrigan's pulse, 305, 313, 363
- Coryza, action of child toward breast in, 24
  - in influenza, 525
- Cough and expectoration, 596
  - causes of night-, 599
  - character of, in acute laryngitis, 598
    - in aneurism of transverse arch of aorta, 307
    - in false croup, 596, 598
    - in laryngeal phthisis, 598

- Cough, chronic loose, morning, in empyema, rupturing into bronchus, 597
    - in pulmonary abscess, 597
    - in sacculated bronchiectasis, 597
    - in tuberculosis with cavity, 597
  - dry and hacking, in phthisis pulmonalis, acute bronchitis, and pneumonia before exudation, 596
  - due to change of position in pleurisy with effusion, 598
  - during aspiration of fluid in pleurisy, 598
  - followed by cry in pneumonia or pleurisy, in children, 24
  - hard and dry, in acute bronchitis, 291, 596
  - hysterical, 599
  - in paralysis of diaphragm, 600
  - in whooping-cough, 596
  - laryngeal, due to irritant dust or vapors, 596, 598
    - due to mediastinal tumors, 598
    - due to pressure by aneurism, 598
      - by carcinoma of œsophagus, 598
  - nervous or reflex, 599
  - obstinate, in Bright's disease, 598
  - produced by enlarged tonsils, enlarged uvula, hypertrophy of mucous membrane of the nose, etc., 599
  - significance of cessation of, in advanced phthisis, bronchorrhœa of the old, and severe pneumonia, 600
  - smothered or suppressed, in pleuro-pulmonary inflammation, 597
  - value of loose, 597
  - varieties of, 596
- Courvoisier's law, 354
- Coxalgia, pain in, 23
- Cracked-pot sound, 264
- Cranio-tabes, 53
- Cremasteric reflex, 626
- Cretinism, facial expression in, 34
- Cretinoid œdema, 210
- Crisis, apparent, in relapsing fever, 513
  - fever in erysipelas ending by either lysis or, 515
  - gastric, in locomotor ataxia, 92, 586
  - in croupous pneumonia, 526
  - in typhus fever, 512
  - intestinal, 453
  - subnormal temperature in many diseases at, 530
  - with fading of eruption in measles, 514
- Crossed anæsthesia, 219
    - in lesion of one side of cord, 220
      - of peduncle, 219
      - of pons, 220
  - diplopia, 469, 472
  - paralysis, 42
- Croup, spasmodic, character of cough in, 596, 598
    - inspirations prolonged in, 252
    - laryngeal spasm in, 294
- Croupous pneumonia. *See* Pneumonia, croupous.
- Crutch-palsy, 76
- Crying in children, absence of, in pneumonia, 24
    - causes of, 24
    - constant, due to earache or hunger, 24
    - of earache, not pacified by offer of breast, 24
  - of child over four months old, without shedding tears, an unfavorable sign, 24
- Crystals, ammonium urate, 426
  - amorphous phosphates, 426
  - blood-, of Teichmann, 413
  - Charcot-Leyden, 603
  - creatin, 426
  - creatinin, 426
  - hæmin, 594
  - leucin and tyrosin, in phosphorus-poisoning, 591
  - oxalate of lime, 425
  - triple phosphates, 426
  - urates, 425
  - uric acid, 425
- Curschmann's spirals in asthmatic attacks, 294, 603
    - rarely in croupous pneumonia, 603
      - in pulmonary tuberculosis, 603
- Curvature of spine, unilateral bulging of chest in, 249
- Cyanosis, causes of, 180, 182
  - due to drugs, as antipyrin or acetanilid, 181
  - in acute articular rheumatism with hyperpyrexia, 528
  - in acute miliary tuberculosis, 525
  - in cardiac dyspnœa, 31
  - in laryngeal obstruction, 181
  - in newborn, 180
    - relative frequency of cause of lesions of, 181
  - in pulmonary diseases, 181
  - in serious cardiac disease, 181
- Cyclic albuminuria, 434
- Cyclical vomiting, other symptoms dependent on, 590
- Cylindroids, significance of, 423
  - varieties of, 422

Cyst, echinococcus, of kidney, 346
    of liver, 353
  hæmaturia in renal, 411
  of kidney, 346
  of mesentery, 346
  of pancreas, 341
  ovarian, diagnosis of, 324
Cystic degeneration of kidney, 346
Cystitis, acute, hæmaturia due to, 411
  character of pain in, 622
  chronic, triple phosphates in urine in, 427
  darting pain in urethra in, 404
  purulent symptoms of septic fever in, 402
  tenesmus in, 404

DACTYLITIS, 59
  syphilitica in infant, 60
Dare's hæmoglobinometer, 378, 379
Debility, night-sweats in, 205
Deformity of feet and legs, 128
    in acute cerebral paralysis of infancy, 110
Degenerative myocarditis, 318
Delirium a bad omen in croupous pneumonia, 280
  in acute articular rheumatism with hyperpyrexia, 528
    yellow atrophy of liver, 552
  in thrombosis of cerebral sinuses, 543
  incoherent speech in, 633
  low, muttering, in typhoid fever, 31, 633
  mild, in mitral regurgitation, 546
  wild, in phosphorus-poisoning and in some cases of uræmia, 592
Dementia, paretic, Argyll-Robertson pupil in, 485
    diplopia in, 466
    gangrene in, 201
    hemiplegia in, 148
    hemorrhage into skin in, 184
    hesitating, halting speech in, 633
    knee-jerk in, 628
    localized sweating in, 205
    optic atrophy in, 497
    perforating ulcer of foot in, 201
    tache cérébrale in, 185, 186
  tremor of tongue in, 633
Dengue, an epidemic disease, 520
  character of fever in, 520
  differential diagnosis of, 521, 522
  erythema in, 186
  hæmatemesis in, 587
  jaundice in, 175
  joint-involvements in, 133
  pain involving entire body in, 624
Dentition, fever in difficult, 505
Dermatitis, acute exfoliating, 187
Diabète bronzé, 174
Diabetes insipidus, constipation in, 449
    great increase of urine in, 408
    loss of knee-jerk in some cases of, 627
    urine of, 447
  mellitus, acetonuria in, 439
    Argyll-Robertson pupil in, 92, 484
    boils in, 199, 438
    breath in, 19
    caries of teeth in, 163
    cataract in, 464, 499
    Cheyne-Stokes respiration in, 252
    color of urine in, 408, 447
    coma in, 447, 551
    constipation in, 449
    development of, in carcinoma of pancreas, 334
    diagnosis of coma in, 551
    dry, harsh skin in, 205, 447
    fermentation of urine in, due to saccharomyces albicans, 430
    furunculosis in, 447
    gangrene in, 201
    great increase of urine in, 408
    headache rarely in, 535
    heavy, sweet odor of urine in, 408
    high specific gravity of urine in, 417
    jaundice in, 174
    knee-jerk lost in advanced, 627
    ocular palsy in, 475
    paraplegia rare in, 124
    perforating ulcer of foot in, 134
    polydipsia in, 438
    polyphagia in, 438
    prognosis of, 438
      if associated with obesity, 438
      influence of age on, 438
    pruritus in, 239, 438
    retinitis in, 499
    roseola in, caused by urine, 192
    rosette crystals of uric acid in urine of, 425
    urine in, 447
    vomiting rare in, 577
    wasting in, 438
    wasting of hands in, 69
    white spots on trousers in, 18
Diagnostic considerations, general, 17
Diaphragm, paralysis of, cough in, 600
    causes of, 600
Diarrhœa, causes of, 450
  colliquative, in uræmia, 577
  decrease of urine due to, 408
  dissecting-room, 457
  fatty, due to cod-liver oil, 457
    due to disease of pancreas, 457
    in jaundice, 457
  in abdominal pain, 23

- Diarrhœa in cholera infantum, 451, 452
  - morbus, 451
  - in dysentery, 455
  - in entero-colitis, 453
  - in fissure of the anus, 453
  - in hysteria, 457
  - in influenza, 525
  - in leukæmia, 388
  - in locomotor ataxia, 453
  - in malignant ulceration, 456
  - in proctitis, 455
  - in pulmonary gangrene or tuberculosis, 456
  - in renal disease, 453
  - in septicæmia, 456
  - in syphilitic ulceration, 456
  - in tuberculosis, 454
  - lienteric, 454
  - may be caused by purgatives, 23
  - nervous, 453
  - pain in, 23
  - paroxysmal, sero-mucous or bloody, in exophthalmic goitre, 457
  - summer, retraction of belly-wall in, 322
- Diazo-reaction, 445
- Dicrotic pulse, 364
- Dietl's crises, 619
- Differential table of symptoms between yellow fever and pernicious malaria and dengue, 522
- Digestion leucocytosis, 386
- Digestive disturbances as a cause of laryngeal spasm, 294
- Dilatation of heart, symptoms and physical signs of, 266, 313
  - of pupil of the eye, 465, 485
  - of stomach, atrophy of gastric tubules in, 337
    - character of vomiting in, 588
    - constitution of vomitus in, 588
    - in pressure by growths of pancreas, 589
    - in tetany, 75
    - methods of examining in, 335, 336
    - sarcinæ ventriculi in vomitus in, 589
    - torula cerevisiæ, causing fermentation in, 589
    - total absence of hydrochloric acid in, 338, 589
- Diphtheria, anæsthesia in 50 per cent. of cases of, due to neuritis, 231
  - casts of larynx and upper bronchial tubes at times coughed up in, 603
  - difficult swallowing due to paralysis from, 164
  - double oculomotor paralysis in, 478
  - nasal, 167
  - paralysis of tongue following, 161
  - paraplegia a sequel to, 124
- Diphtheria, roseola in, 189
  - sickening sweet odor of breath in, 19
  - symptoms of, 166, 167
- Diphthertic dysentery, secondary, 456
  - paralysis, dysphagia due to, 164
- Diplegia, spastic congenital, 104
- Diplopia, crossed, 469, 472
  - explanation of. 466
  - homonymous, 469, 472, 474
  - in meningitis, 466
  - in poisoning, 466
  - symptom of Friedreich's ataxia, 466
    - of lesion at base of brain, 466
      - of cerebral cortex, 466
      - of cranial nerve nuclei, 466
      - of nerve in its course, 466
    - of locomotor ataxia, 466
    - of paretic dementia, 466
  - vertical, 474
- Direct percussion, 258
- Dislocation, spontaneous, of the hip following infectious diseases, 134
- Dissecting glossitis, 156
- Distoma hæmatobium, eggs of, in urine, 428
    - hæmaturia due to, 412
  - pulmonum, 605
- Dittrich's plugs, 292
- Diverticulum of œsophagus, 165
- Dropsy, 206. *See* Œdema and general anasarca.
  - general, in arsenic-poisoning, 207
    - in beri-beri, 207
    - in blood diseases, 206
    - in cancerous cachexia, 207
    - in disordered nervous control of vessels, 206
    - in emboli, 206
    - in heart disease, 206, 207, 312
    - in multiple neuritis, 207
    - in renal disease, 206
    - in scurvy, 207
    - in thrombi, 206
    - in vascular disease, 206
  - localized, in anæmia, 207
    - in aneurism, 209
    - in angioneurotic œdema, 209
    - in arsenic-poisoning, 208
    - in cardiac failure, 207
    - in cerebral thrombosis, 209
    - in hepatitis or cirrhosis, 207
    - in mastoid abscess, 209
    - in neuralgia, 209
    - in occupations requiring a standing position, 207, 208
    - in phlegmasia alba dolens, 208
    - in pressure on great vein, 207
    - in pyothorax, 209
    - in relapsing fever, 209
    - in renal disease, 207
    - in thrombosis, 209
    - in typhoid fever, 208, 209

- Dropsy, localized, of eyelids in renal disease, 208
    - in other affections, 209
  - of feet and legs in abdominal growths causing pressure, 207
    - in anæmia, 208
    - in cancer of pancreas, 207
    - in renal disease, 207
- Dryness of the skin, 205
- Dry râles, 274
- Dubini's chorea, 78, 573
- Dupuytren's contraction, 63
- Dysentery, acute primary, of a diphtheritic character, 456
  - amœbic, amœba coli the cause of, 455
    - liver abscess in, 456
    - symptoms of, 455
  - arthritis in, 133
  - character of stools in, 455
  - epidemic, due to Shiga's bacillus, 456
  - fever in, 455
  - secondary diphtheritic, a complication of Bright's disease, 456
    - in acute croupous pneumonia, 456
    - in chronic heart disease, 456
  - slight chill in, 455
  - thirst in, 455
  - tongue in, 154
  - tropical, 455
  - vomiting in, 586
- Dysmenorrhœa, pain in, 622
- Dysphagia, causes of, 164
- Dyspnœa from foreign body in air-passages, 294
  - in anæmia, 384
  - in aortic insufficiency, 312
  - in asthma, 293
  - in chlorosis, 385
  - in heart disease, 31
  - in laryngeal spasms, 294
  - in leukæmia, 388
  - in miliary tuberculosis, 579
  - in pneumothorax, 291
  - inspection of chest in, 252
  - position due to, 21
  - worse at night in chronic interstitial nephritis, 447

- EARACHE, character of crying in children with, 24
  - rubbing of hand over affected side of head in, 24
- Echinococcus cysts of kidney, 346
  - enlargement of gall-bladder due to, 353
  - of liver, jaundice in, 174
- Echolalia, 49, 78
- Eclampsia, puerperal. *See* Puerperal eclampsia.
- Eczema, appearance of nails in, 59
  - due to chloral, 199
  - due to mercury, 199
  - due to potassium iodide, 199
  - due to quinine, 199
- Effusion, pleural, bronchial breathing in, 273
  - decreased vocal fremitus in, 256
  - displaced apex-beat in, 254, 255
  - flat percussion-note over effusion, 264
  - hemorrhagic, 290
  - serous, Pohl and Rosenbach's test in, 290
    - sugar in, 326
    - thrombosis of vena azygos as a cause of, 290
  - sero-purulent, absence of sugar in, 326
  - skodaic resonance in, 263
- Ehrlich, corpuscles of, 384
  - diazo-reaction of, in urine, 445
  - myelocyte of, 387
  - triple blood-stain of, 382
- Electric chorea, 78, 573
    - Bergeron's, 78
- Elephantiasis sometimes caused by filaria sanguinis hominis, 398
  - symptoms of, 210
- Embolism, cerebral, brachial monoplegia in, 85
    - Cheyne-Stokes respiration in, 252
    - gangrene in, 135
    - vomiting in, 577
  - of coronary arteries, 318
  - of kidney, casts of, micrococci in, 421
    - hæmaturia in, 409, 411
    - œdema in, 206
  - of pons Varolii, 528
  - of superior mesenteric artery, symptoms of, 591
- Emphysema, appearance of hands in, 58
  - barrel-shaped chest of, 246, 292
  - distended cervical vessels in, 253
  - expiration prolonged in, 252, 292
  - lessened area of cardiac dulness in, 269, 292
  - percussion-resonance increased in, 292
  - pulmonary and cardiac hypertrophy causing depression of apex-beat in, 255, 256
  - râles in, 292
  - spleen and liver displaced downward in, 293, 358
  - systolic tricuspid murmur in, 292
  - vocal fremitus diminished in, 292
    - resonance diminished in, 278, 292
- Emprosthotonos in hysteria, 565

- Empyema communicating with bronchus, loose morning cough in, 597
  - in children, 289
  - odor of breath in, 19
  - organisms causing, 290
  - peptonuria in, 435
  - pulsation of chest-wall in some cases of, 258
  - purulent sputum in, 602
  - symptoms of, 289
- Endarteritis, syphilitic, immobility of pupil in, 485
- Endocarditis, acute ulcerative, character of fever in, 511, 517
    - diagnosis between typhoid fever and, 511
    - duration of, 517
    - hæmaturia in, 409
    - jaundice in, 177
    - petechial rashes in, 184
    - prognosis of, 519
    - purpuric eruptions in, 184
    - retinal hemorrhage in, 499
    - sweating in, 204
- Enteric fever. *See* Typhoid fever.
- Enterocolitis, flakes of mucus in stools of, 454
  - mild follicular, 454
  - ribbon-shaped stools in, 454
  - symptoms of, 453, 454
  - ulcerative, 454
- Enteroliths, 460, 584
- Eosinophile corpuscles, 382
    - in myelogenous leukæmia, 382
    - in trichinosis, 382
- Epidemic cerebro-spinal meninigitis, coma in, 554
  - dysentery due to Shiga's bacillus, 456
- Epigastrium, distention of, 328
  - pulsation in region of, 255, 343
- Epilepsy, alcoholic, 561
  - aura in, 556
  - biting of tongue in, 157, 557
  - caused by drugs, 561
    - by lead, 561
    - by malaria, 563
  - character of convulsions in, 556
  - cutaneous hemorrhage in, 184
  - diagnosis between attacks simulated by malingerers and, 569
    - between hysteria and, 564
      - puerperal eclampsia and, 568
      - syncope and, 569
      - uræmia and, 567
  - expression of face in, 556, 557
  - facial spasm in, 48, 556
  - fever after seizure of, 529, 567
  - hippus in, 186
  - Jacksonian, character of convulsions in, 557, 558
  - knee-jerk in, 627
- Epilepsy, minor, diagnosis of, 568
    - unconsciousness in, 555
    - vertigo the only symptom of, 547
  - muscæ volitantes in, 499
  - nocturnal, headache due to, 546
  - nystagmus, a rare symptom of, 482
  - post-hemiplegic, 558
  - scars on head in suspected traumatic, 204
  - spasm of heart in, 555
  - syphilitic, 560
    - diagnosis of, 560, 561
    - symptoms of, 560
  - *tache cérébrale* in, 185
  - vertigo, a premonitory symptom of, 547
- Epileptiform convulsions from cerebral hemorrhage, 558
    - from other causes, 559
    - in cerebral palsy of children, 74
- Epiphysitis of infancy, acute symptoms of, 132
- Epithelial casts in urine, 419
- Epithelioma, ulceration of tongue in, 156
- Equina. *See* Glanders.
- Erb's paralysis, 87
  - symptom, 571
- Ergotism, gangrene in, 67, 202
  - petechiæ in, 184
- Eruption, date of, in various diseases, 189
  - herpetic, along nerve-trunk in sciatic neuritis, 615
  - in anthrax maligna, 199
    - simplex, 199
  - in chickenpox, 194, 197
  - in erysipelas, 191, 200
  - in glanders, 200
  - in impetigo contagiosa, 198
  - in measles, 188
  - in rheumatism, 182
  - in rötheln, 188
  - in smallpox, 189, 194, 514
  - in syphilis, 203
  - in typhus, 191, 512
  - in vaccinia, 196
  - of skin in regard to age, 182
  - pemphigus-like, due to salicylic acid or copaiba, 200
  - pharyngeal and buccal, in measles, 167
  - purpuric, from diseases, 183
    - from drugs, 184
  - resulting from quinine, 199
  - vesicular, about mouth, in foot-and-mouth disease in man, 159
- Erysipelas, appearance of eruption in, 191, 200
  - chill in, 501
  - diazo-reaction in severe, 445
  - fever in, ending by either crisis or lysis, 515

- Erysipelas, fever in, marked remissions and intermissions of, 515
    - sudden rise of, 515
  - inflammation of skin similar to, from arnica, 191
  - œdema of face in, 32
  - peptonuria in, 435
  - symptoms of, 191
  - vomiting in, 594
- Erythema. *See* also Roseola.
  - exudativum multiforme, symptoms of, 185
  - following operation, 190
    - parturition, 190
    - vaccination, 189
  - in Bright's disease, 191
  - in dengue, 186
  - in diabetes mellitus, 447
  - in diphtheria, 189
  - in rheumatism, 182
  - in scarlet fever, 186
  - in smallpox, 189
  - in syphilis, 190
  - in typhoid fever, 191
  - produced by drugs, 192, 198
  - roseola, symptoms of, 187
  - scarlatiniform, symptoms of, 187
- Erythrocytes, 367
- Esbach's test for albumin in urine, 433
- Esophoria, 467
- Ewald's test-breakfast, 339
- Examination, method of, in consulting-room, 27
  - of abdomen, 321
  - of blood by hæmatocrit, 375
    - by hæmatocytometer, 370
    - by hæmoglobinometer, 376
    - for micro-organisms of malaria, 396
    - in leukæmia, 386
  - of children, 23
  - of eye by ophthalmoscope, 493
  - of field of vision, 490
  - of functional activity of ocular muscles, 468
  - of hemianopsia, 491
  - of pulse, 359
  - of pupillary reflex, 483
  - of skin for jaundice, 170
  - of sputum, 600, 604
  - of stomach, 332
  - of tongue, 150
- Exanthemata, date of eruption of various, 189
- Exhaustion from hiccough, 576
- Exophoria, 467
- Exophthalmic goitre. *See* Goitre, exophthalmic.
- Exophthalmus in thrombosis of cavernous sinus, 543
- Expectoration. *See* Sputum.
- Expression of face as an early symptom of facio-humero-scapular type of muscular atrophy, 33
  - elated, of paretic dementia, 32
  - excited, of acute mania, 32
  - fatuous, of hysteria, 32
  - fixed, of catalepsy, 32
  - how formed, 27
  - how modified, 27
  - in acute fever, 31
    - peritonitis, 32
    - pulmonary phthisis, 31
  - in alcoholics, 28
  - in cretinism, 34
  - in croupous pneumonia, severe, 31
  - in dyspnœa of heart disease, 31
  - in epileptic seizures, 556
  - in Friedreich's ataxia, 33
  - in healthy sleeping child, 29
  - in hysteria, 32
  - in malignant disease, 29
  - in melancholia, 32
  - in moderate pain in children, 30
  - in myxœdema, 34, 37
  - in nervous exhaustion, 32
  - in renal disease, 32
  - in typhoid fever, 31
  - intellectual, 27
  - of anxiety in grave disease, 29
  - of "mouth-breathers," 30
- Eye, ability to close, in cerebral facial paralysis, 39
  - alteration of color-field of, 492
  - amaurosis, 500
  - anatomy of optic nerve of, 487
  - annulus senilis, 465
  - appearance of healthy eye-ground, 495
  - arcus senilis, 465
  - causes of paralysis of, 469, 475
  - conjunctival hemorrhage, from coughing, 464
    - from degenerative vascular changes, 464
    - from injury, 464
  - dark areas under, during menstruation, 28
  - diagnosis between ocular symptoms of tabes and hysteria, 493
  - diplopia, 466
  - examination of, ophthalmoscopic, 493
  - exophthalmos in goitre, 464
    - in lesion of oculomotor nerve, 43
  - Graefe's symptom, 465
  - hemianopsia, 487
  - homonymous and crossed diplopia, 469
  - intraocular muscles of, 482
  - iritis, 498, 499

Eye, lesion of optic nerve in locomotor ataxia, 497
method of determining paralysis of, 470
of examining functional activity of muscles, 468
muscæ volitantes, 499
orthophoria, 468
papillitis, 496
paralysis of muscles of, 467
puffiness about, from excessive use of arsenic, 28
in acute diffuse nephritis, 446
retinitis, 498
retrobulbar neuritis, 497
rod-test of Maddox, 468
squint, 467
-strain, headaches due to, 533
Eyelids closed in healthy sleeping child, 29
œdematous, in angioneurotic œdema, 209
from arsenic, 208
in cerebral thrombosis, 209
in neurotics, 209
painful twitchings of, accompanying facial hemiatrophy, 37
pallor of, in anæmia, 464
pigmentation of, early in pregnancy, 28
puffy, in cardiac disease, 464
in overuse of arsenic, 464
in renal disease, 28, 464
in trichinosis, 32
showing glazed conjunctivæ, 30
slightly parted in sleeping child, showing congestive or nervous pain, 29
swollen in cretinism, 34
twitching in nervous irritation, 30

FACE. *See* also Expression.
anæsthesia of, due to involvement of the fifth nerve or its nucleus, 234
anxious and covered with sweat in angina pectoris, 613
asymmetry of, congenital, 35
blurring of features of, in children with lesions of mitral valve, 31
diagnosis of anæsthesia of, 234, 235
facio-humero-scapular type of muscular atrophy, 33
full-moon, of myxœdema, 34, 38
gray or bluish, from overdose of coal-tar products, 28
heavy, cheesy-looking, in children, 30
hemiatrophy of, 35
hemihypertrophy of, 37
Hippocratic, of impending death, 35
hysterical, 28, 32, 565, 566
intellectual, 27
Face, leonine, of leprosy, 34
massive, of acromegaly, 35, 38
œdema or great swelling of, in dropsy, 32
in erysipelas, 32
of acute peritonitis, 31
of carcinoma, 29
of catalepsy, 32
of chorea in children, 32
of chronic or subacute renal disease, 32
of congenital syphilis, 30
of cretinism, 34
of disseminated sclerosis, early stages of, 32
of Friedreich's ataxia, 33
of leprosy, 34
of paralysis agitans, 32
of pneumonia, severe, 31
of rickets, 30
of those exposed to weather, 28
living indoors, 28
using alcohol in excess, 28
of typhoid fever, 31
pallor and puffiness of, in leukæmia, 388
pallor of, in chlorosis, 28, 180, 385
in fright, 28
in hemorrhage, 28
paralysis of, bilateral, 46
unilateral, 38
parchment-like skin of, in syphilis and alcoholic hepatic cirrhosis, 28
pellucid, in renal disease in children, 32
puffiness of, about eyes, in acute diffuse nephritis, 446
spasm of, 48
triangular, in osteitis deformans, 38
Facial deformity, 35
expression from nasal obstruction, 30
Family idiocy, amaurotic, 104
periodic paralysis, 125
Farrant's solution, 424
Fat, masses of. *See* Adiposis dolorosa.
necrosis, from impacted gallstone, 341
Fatty casts in urine, 422
degeneration in phosophorus-poisoning, 592
heart, 318
Feces. *See* also Stools.
causes of variation in quantity of, 457
color of, 458
consistency of, 458
impaction of, 343
pain in, 620
odor of, 458
Feet and legs, deformities of, 128
claw-, 108
contractures of, in hysteria, 111

Feet, deformity of, due to acute cerebral paralysis of infancy, 110
    due to poliomyelitis, 127
    due to progressive muscular atrophy, 129
  distribution of anæsthesia of, in neuritis, 234
  enlarged, due to deformity, 129
    in acromegaly, 128
    in myxœdema, 128
    in pulmonary osteo-arthropathy, 128
  flat-, in locomotor ataxia, 129
  numbness of, in locomotor ataxia, 92
  perforating ulcer of, in diabetes, 134
      in senile gangrene, 134
      in tabes dorsalis, 134
  sciopedy, 129
  tabetic, 129
Fehling's test for sugar in the urine, 436
Femur, malignant growth of, diagnosis of, from sciatica, 616
Festination, 100, 573
Fever, 501
  absence of, in pulmonary œdema, 286
  appearance of nails in, 59
  causes of tertian, quartan, and æstivo-autumnal, 390–392
  cold, wet skin of evil import in, 504
  character of, in acute appendicitis, 528
      articular rheumatism, 528
      miliary tuberculosis, 525
      multiple neuritis, 529
      myelitis, 529
      pulmonary tuberculosis, 524
    in anthrax, 527
    in catarrhal pneumonia, 526
    in cerebral abscess, 524
    in cerebro-spinal meningitis, 522
    in chickenpox, 515
    in cholangitis, catarrhal or suppurative, 354, 519
    in cholera Asiatica, 527
      infantum, 527
    in croupous pneumonia, 525
    in dengue, 133, 520
    in difficult dentition, 505
    in dysentery, 455
    in epilepsy, 529, 567
    in erysipelas, 515
    in foot-and-mouth disease, 527
    in gastro-pulmonary fever, 530
    in hæmoglobinuria, 593
    in hay-fever, 525
    in hepatic abscess, 519
    in Hodgkin's disease, 519
    in hysteria, 529
    in infantile spinal paralysis, 529
    in influenza, 525
    in injuries to cervical portion of spinal cord, 529
Fever, character of, in intermittent malarial fever, 515
    in Malta fever, 512
    in measles, 514
    in mild gastro-intestinal catarrh in children, 505
    in obstruction of the rectum, 585
    in parenchymatous nephritis, 527
    in pemphigus, 200
    in pernicious anæmia, 519
    in pyelitis, 448, 520
    in relapsing fever, 512
    in remittent malarial fever, 520
    in rötheln, 514
    in scarlet-fever, 513
    in septic poisoning, 519
    in septicæmia, 524
    in smallpox, 514
    in syphilis, 526
    in teething, 505
    in tetanus, 528
    in thermic fever, 528
    in tonsillitis, 527
    in trichinosis, 512
    in typhoid fever, 506
    in typhus fever, 512
    in ulcerative endocarditis, 511, 517
    in Weil's disease, 520
    in yellow fever, 521
  Charcot's, 175
  chlorides decreased in some, 445
    increased during convalescence from, 445
  definition of, 501
  dry, hot skin in, 504
  flushed face in, 31, 504
  high specific gravity of urine in, 417
  infantile remittent, 520
  rapid pulse in, 366
    respiration in, 251
  secondary, in smallpox, 194
  septic, in purulent cystitis, 402
  sighing or arhythmic respiration in, in children, 25
  significance of, 504
    in children, 505
  slight, in chlorosis, 385
    in leukæmia, 388
  urates in excess in urine of, 425
  urea increased in urine of, 444
  urethral, from passing sounds, 505
  uric acid increased in urine of, 425
  urine decreased in, 408
Fibrinous bronchitis, small casts of bronchial tubes in sputum in, 604
Fibroid phthisis, inspection of chest in, 284
Filaria Demarquayi, 398
  description of, 398

Filaria diurna, 398
  embryos of, in urine, 412, 428
  hæmaturia in, 412
  loa, 398
  nocturna, 398
  Ozzendi, 398
  perstans, 398
  sanguinis homonis, chyluria, and elephantiasis caused by, 400
Fingers, appearance of, diagnostic of chloral-habit, 60
  clubbed in heart disease, 58, 312
  distorted and twisted in gout and arthritis deformans, 59
  fixation of joints of, by deposit of urate of sodium, 63
  in syphilitic dactylitis, 59
  spasm of, due to occupation, 76
"Fish-mouth" of nasal obstruction, 30
Fissure of the anus, character of pain in, 623
    diarrhœa in, 453
Flat-foot in locomotor ataxia, 129
Flint's murmur, 304
Floating kidney, 343
    pain in, 619
  liver, 352
  spleen, 346
Fluoroscope in diagnosis of aortic aneurism, 309
    of tuberculosis, 285
Follicular enterocolitis, mild, 454
  tonsillitis, symptoms of, 166
Fontanelle, condition of, diagnostic, 52–55
Foot-and-mouth disease in man, symptoms of, 527
Foot-drop, 126
Forehead, immense and bulging, with a wizened, puny face beneath, indicating hydrocephalic tendencies, 30
  square and projecting, indicative of rickets, 30
  wrinkled, indicating pain in the head, 30
Foreign body in air-passages, 294
    in bowel, 343
Formication, 216
"Fourth disease" of Dukes, 187
Fovea centralis, 496
Fremitus, vocal, decrease of, causes of, 256
    increase of, causes of, 256
    mode of production of, 255
Friction-sounds, 276
  as a rule, due to pleuritis, 276
  at apex of chest, usually due to tuberculosis, 277
  best heard in axilla, 276
  pericardial, 276, 311
Friedreich's ataxia, claw-foot in, 109
Friedreich's ataxia, diagnosis of, 628
    differential points between locomotor ataxia and, 95, 96
    diplopia a symptom of, 466
    face of, 33
    gait of, 95
    loss of knee-jerk in, 627
    movement of hands in, 82
    nystagmus in, 482
    sensory disturbance of skin late in, 222
    slow, scanning speech in, 633
    table of symptoms of, 95, 96
Frog-belly, 322
Frost-bite, gangrene in, 67
Fungus foot disease. *See* Mycetoma.
Furunculosis in diabetes mellitus, 447
Futcher's method of staining malarial parasites, 397

GABBETT'S method of staining for tubercle bacilli, 608
Gaertner's tonometer, 362
Gait, 90
  "astasia abasia," 101
  in acute poliomyelitis, 98
  in cerebellar disease, 101
    tumor, 101, 537
  in chronic myelitis, 97
  in disseminated sclerosis, 96, 97, 99
  in Friedreich's ataxia, 95, 96
  in general paresis, 94
  in gout, 90
  in health, 18
  in hemiplegia, 101
  in hereditary cerebellar ataxia, 96, 97
  in Huntingdon's chorea, 573
  in hysteria, 101
  in infantile cerebral paralysis, 99
  in lateral sclerosis, 97, 98
  in locomotor ataxia, 91, 94
      cause of, 92
  in multiple sclerosis, 97, 100
  in neurasthenia, 18
  in osteomalacia, 102
  in paralysis agitans, 100
  in pseudo-muscular hypertrophy, 98
    -tabes, 91
  in rheumatism, 90
  in rickets, 99, 102
  in sciatica, 90
Gall-bladder, cancer of, 355
  diagnosis of obstruction of, 353
Gallop-rhythm in mitral stenosis, 303
Gallstone colic, 341, 354, 619
  in stools, 459
  jaundice in cases of, 172
Gallstones, enlargement of gall-bladder due to, 353
Gametocytes, 394
Gangrene in central nervous lesion, 201

Gangrene in diabetes, 201
  in embolism, 135
  in ergotism, 67
  in exophthalmic goitre, 135
  in frost-bite, 67
  in leprosy, 67
  in nerve injury, 201
  in Raynaud's disease, 67, 201
  of extremities, following infectious fevers, 135
    in diabetes mellitus, 447
  of intestines, fetid odor of stools in, 458
  pulmonary, character of sputum in, 286, 601, 602
    crystals of margaric acid in sputum of, 605
    due to putrid bronchitis, 292
    elastic fibres in sputum of, 605
    fetid breath in, 286
    muco-purulent stools in, 456
    vomiting in, 594
  senile, perforating ulcer of foot in, 134
  spontaneous, in hysteria, 201
Gastralgia, character of pain in, 622
  diagnosis of, 622
  neuralgic spots in, 622
  position in, 20
Gastric cancer, anæmia in, 385
    fever in, 519
    indicanuria in, 415
    most frequent at pylorus, 332
    vomiting in, 332, 586
  catarrh, acute, symptoms of, 588
      tongue in, 153
      vomiting in, 588
    chronic, symptoms of, 588
      vomiting in, 588
  crisis in locomotor ataxia, 92
  dilatation, 334. *See* Dilatation of the stomach.
  disorders, odor of breath in, 19
  ulcer, anæmia in, 334, 385
    pain in, 334, 614
    symptoms of, 333, 586
    vomiting of blood in, 586
Gastritis, vomiting rare in, excepting when due to an irritant, 588
Gastro-diaphane of Einhorn, 336
  -duodenal catarrh, jaundice in, 170
  -pulmonary fever, etiology of, 530
      gastric symptoms of, 530
      marked fever in, 530
      pulmonary symptoms of, 530
Gastroptosis, Glenard's belt sign of, 337
  jaundice as result of, 174
General anasarca in acute diffuse nephritis, 446
    in advanced cancerous cachexia, 207
    in arsenic-poisoning, 207
    in beri-beri, 207
    in children with acute diffuse nephritis, 446
    in chronic parenchymatous nephritis, 446
    in heart disease, 206, 207
    in multiple peripheral neuritis, 207
    in renal disease, 206
  diagnostic considerations, 17
  paresis. *See* Paresis, general.
Geographical tongue, 159
Gerhardt's symptom, 544
German measles. *See* Rötheln.
Girdle-sensation, 237
  in locomotor ataxia, 237
  in myelitis, 106, 237
  in tumors of cord and meninges, 237
Glanders, eruption of, 200
  symptoms of, 200
Glaucoma, dilated pupil in, 465, 486
  headache in, 534
Glenard's belt sign of gastroptosis, 337
Globus hystericus in hysteria, 566
Glossitis, acute, 159
  chronic superficial, 158
  dissecting, 158
Glossodynia exfoliativa, 158
Glosso-labio-pharyngeal paralysis, 46
    bilateral atrophy of tongue in, 159
    cardiac feebleness in, 318
    dysphagia in, 164
    movements of tongue in, 160
    tremor of tongue in, 162
Glycosuria, Fehling's test for, 436
  Haines's test for, 435
  phenyl-hydrazin test for, 437
  Roberts's yeast-test for, 438
  significance of, 438
  Trommer's test for, 437
  Whitney's test for, 435
Gmelin's test for bile in urine, 414, 440
Goitre, exophthalmic, Abadie's sign in, 48, 55, 56
    brown pigmentation an occasional symptom of, 179
    diminished electrical resistance in, 317
    exophthalmos in, 56, 464
    gangrene in, 135
    Graefe's symptom of, 465
    localized sweating in, 205
    Moebius's sign of, 465
    paroxysmal bloody, mucous diarrhœa in, 457
    Stellwag's symptom in, 465
    symptoms of, 464
    tache cérébrale in some cases of, 185

Goitre, exophthalmic, tachycardia in, 56, 317
    thrill over carotid arteries in, 317
    tremor of hand in, 81
    vomiting in, 592
Gonococci as a cause of empyema, 290
  indicative of specific urethritis or vaginitis, 428
  method of staining, 428
Gonorrhœal arthritis, symptoms of, 130
Gout, aortitis in, 613
  appearance of nails in, 59
  distortion of fingers in, 59
  hyperæsthesia of scalp in, 238
  joint-affections in, 132
  limping gait of, 90
  pain in great toe in, 624
  plumbic, 133
  pruritus in, 239
  retinal hemorrhages in, 499
  urates in urine of, 425
  uric acid in urine of, 425
Gouty diathesis, appearance of nails in, 59
Graefe's spots, 48
  symptom, 465
Granular casts in urine, 421
Grave's disease, tremor of hand in, 81
Gravid leucocytosis, 386
"Grisolle" sign of smallpox, 189
Guaiacum test for hæmoglobin in urine, 413
Gums, blue line on, in lead-poisoning, 163
  spongy, in salivation, 164
    in scurvy, 164
Gyromele of Türck, 336

HABIT-CHOREA, 77
  -spasm, 48, 77
Hæmatocrit, 375
Hæmatoma auris, 184
Hæmin, test for, 594
Hæmoglobin, 367
  diagnosis of syphilis by studying the state of, 386
  in chlorosis, 385
  in leukæmia, 386
  specific-gravity method of estimating, 379
  Tallquist method of estimating, 381
Hæmoglobinometer, Dare's, 378
  Von Fleischl's, 376
Haines's modification of Haeser's method for computing total quantity of solids in urine, 417
  test for sugar in urine, 435
Hands, "accoucheur's," in tetany, 75
  changes in shape of, in amyotrophic lateral sclerosis, 73
      in chronic rheumatism, 65
Hands, choreic movements of, and of arms, in children with chorea minor, 77
  claw-, 68, 69, 70
    in Morvan's disease, 72
  cold and clammy, due to bromidrosis, 60
      due to local innervation of sweat-glands, 60
  contractions of, following apoplexy, 74
    from hysteria, 74
  distribution of anæsthesia of, in neuritis, 233, 234
  Dupuytren's contraction of, 63
  excessive sweating of, in progressive muscular atrophy, 60
  flexion of, in cerebral palsy of children, 73
  gangrene of, in ergotism, 67
    in frost-bite, 67
    in leprosy, 67
    in Morvan's disease, 68
    in neuritis, 68
    in Raynaud's disease, 67
    in syringomyelia, 68
    senile, 67
  in acute articular rheumatism, 66
  in angioneurotic œdema, 67
  in child with cerebral trouble, 24
    with heart disease, 58
  in emphysema and chronic phthisis, 58
  in heart disease, 20
  in thoracic aneurism, 58
  lateral drop of, in neuritis or acute infantile poliomyelitis, 76
  movement of, 78, 81
  position of, in meningeal congestion or hydrocephalus in children, 75
  seal-fin, in arthritis deformans and gout, 65
  spade-like, in acromegaly, 68
    in myxœdema, 68
    in pulmonary osteo-arthropathy, 68
  spasm of, from cold and exposure, 79
    in ataxia, 79
    in paralysis agitans, 79
    in Thomsen's disease, 79
  swelling and rupture of, in mycetoma, 68
  tremors of, 79
  wasting of, in diabetes mellitus, 69
    in nervous lesions, 69
    in old age, 69
    in phthisis, 69
    with anæsthesia, in syringomyelia, 73
    with flexion and rigidity rarely seen in paralysis agitans, 73
Harrison's grooves in rickets, 247

- Haut mal, 568
- Hay fever, 525
- Haygarth's nodosities, 65
- Head, activity of, with fixation of body, in peritonitis, 20
  - examination of, 50
  - excessive sweating of, 56
  - in acromegaly, 52
  - in cholera infantum, 452
  - in chorea minor, 50
  - in cretinism, 52
  - in hydrocephalus, 52
  - in idiocy, 52
  - in microcephalus, 52
  - in myxœdema, 52
  - in osteitis deformans, 52
  - in rickets, 52, 55
  - neuralgia of, 610
  - position of, in advanced strabismus, 52, 470
    - in chronic deafness, 52
    - in mental depression, 52
  - retraction of, in attack of indigestion in neurotic babies, 51
    - generally due to basal meningitis, 51
    - may be due to caries of cervical vertebræ, 51
    - may be due to cerebro-spinal fever in adults, 51
    - may be due to a fall, 52
    - may be due to tender and enlarged glands of neck, 51
  - tremor in disseminated sclerosis, 80
- Headache, causes of, 531
  - due to hemorrhage, 546
  - from use of drugs, 535
  - in acute parenchymatous nephritis, 527
  - in Addison's disease, 593
  - in anæmia, 546
  - in anterior poliomyelitis, 529
  - in auto-intoxication, 532, 533
  - in brain abscess, 539
    - tumor, 536, 578
  - in cerebral tumors, 578
  - in cerebro-spinal meningitis, 522
  - in croupous pneumonia, early stages, 545
  - in dental caries, 546, 547
  - in diabetes, 535
  - in digestive disturbances, 531
  - in disease of cranial bones, 546
  - in ear disease, 547
  - in exposure to cold, 546
  - in eye-strain, 531, 533
  - in gastro-pulmonary fever, 530
  - in glaucoma, 534
  - in hæmatoma of the dura, 545
  - in hæmoglobinæmia, 533
  - in intermittent fever, 546
  - in intracranial aneurism, 546
- Headache in jaundice, 533
  - in malaria, 546
  - in Malta fever, 512
  - in measles, 545
  - in meningeal hemorrhage, 545
    - tumors, 536
  - in meningitis, 540
  - in migraine, 532
  - in nasal catarrh, 546
  - in neurasthenia, 534
  - in nocturnal epilepsy, 546
  - in paroxysmal hæmoglobinuria, 533
  - in phosphaturia, 535
  - in phosphorus-poisoning, 591
  - in renal disease, 535
  - in rheumatism, 535
  - in smallpox, early stages, 545
  - in sunstroke, 545
  - in syphilitic arteritis, 538
    - epilepsy, 560
    - gumma, 538
    - meningitis, 538
  - in tubercular meningitis, 540
  - in typhoid fever, 545
  - in typhus fever, 512
  - in uræmia, 535
  - in valvular heart disease, 546
  - vomiting in, 593
- Heart, alterations in area of dulness of, 255
  - apex-beat of, area of, 256, 257
    - depression of, due to pulmonary emphysema, pericardial or pleural effusion, tumors in the chest, and cardiac hypertrophy, 256
    - displacement of, to left, by hypertrophy of left ventricle, 257
      - by pleural adhesions, 257
      - by pleural effusions, 256
      - to right, by hypertrophy or dilatation of right ventricle, 257
    - normal position of, 256
    - raised by ascites, 257
      - by distention of colon, 257
      - by enlargement of spleen, 257
      - by hypertrophy of heart, 257
      - by intra-abdominal growths, 257
    - strength of, 257
  - asthma due to lesions of, 294
  - blood-streaked sputum in valvular lesions of, 601
  - causes of pain in neighborhood of, 613
  - congenital malformations of the, 319
    - rules for diagnosis of, 319

- Heart, cough in valvular disease of, 599
  - dilatation of, 257, 266, 313
  - disease of, appearance of hands in children in, 58
    - jaundice in chronic valvular, 173
    - œdema in, 207
    - patient's position in, 20, 21
    - retinitis in, 498
  - -failure, in influenza, 316, 525
    - unconsciousness in, 555
  - fatty degeneration of, 318
    - deposition on, 318
  - general malformation of, 319
  - greatly hypertrophied, in chronic interstitial nephritis, 447
  - headache in valvular disease of, 546
  - hypertrophy of, 314
  - location of murmurs of, 300
  - murmurs, significance of, 299
  - neuroses of, 316
  - percussion of, 264
  - secondary diphtheritic dysentery in chronic disease of, 456
  - slightly dilated, in acute diffuse nephritis, 447
    - hypertrophied, in chronic parenchymatous nephritis, 447
  - -sounds, 297
    - accentuation of, 298
      - in chronic contracted kidney, 298
      - in general arterial sclerosis, 298
      - pulmonary second, in mitral obstruction and regurgitation, 298
    - normal, 297
    - reduplication of, 299
    - where best heard, 298
  - symptoms associated with murmurs of, 311
  - thrills of, 258
    - causes of, 258
  - tobacco-, 316
  - valvular lesions of, symptoms of, 311
- Heat-exhaustion, subnormal temperature in, 530
  - -stroke. *See* Sunstroke.
- Heberden's nodes, 65
- Heel and toe, tender, in sciatica, 616
- Heller's test for albumin in urine, 431
  - for blood in urine, 413
- Hematemesis, as vicarious menstruation, 587
  - causes of, 587
  - diagnosis between hæmoptysis and, 587
  - diseases in which it occurs, 587
  - due to swallowed blood, 587
  - in cirrhosis of liver, 587
  - in embolism in the superior mesenteric artery, 591
- Hematemesis in gastric cancer, 586
    - ulcer, 586
  - in heart disease, 587
  - in injury of stomach, 587
  - in locomotor ataxia, 586
  - in malaria, 587
  - in malingerers, 587
  - in melæna neonatorum, 587
  - in scurvy, 587
- Hematocrit, 375
- Hematocytometer of Thoma-Zeiss, 370
- Hematogenous jaundice, 175
- Hematoma auris, 184
  - of dura, headache in, 545
    - mater, convulsions due to, 572
  - subperiosteal, in children, 132
- Hematozoa malariæ, 389
- Hematuria, acute, due to embolism, 409
    - due to irritating drugs, 409
    - due to malaria, 409, 410
    - due to scarlet fever, 409
  - blood-casts in, 420
  - character of blood from bladder in, 409
      - from kidney in, 409
      - from urethra in, 409
  - chronic, due to chronic hemorrhagic nephritis, 410
    - due to cyst of kidney, 411
    - due to distoma hæmatobium, 412
    - due to filaria sanguinis hominis, 412
    - due to renal cancer, 410
    - due to stone in kidney, 411
    - due to strongylus gigas, 412
    - due to tuberculosis of kidney, 411
    - other causes for, 412
- Hemiachromatopsia, 493
- Hemianæsthesia due to lesion of optic thalamus, 218
  - in apoplexy, 218
  - in capsular disease, 216, 218
  - in chorea, 217
  - in cortical lesions, 218
  - in disseminated sclerosis, 218
  - in hysteria, 216
    - superimposed in infantile cerebral paralysis, 217
  - in softening of brain, 218
  - in tumor of brain, 218
  - partial, with partial hemiplegia on opposite side, may be due to lesions of one side of cord, 220
- Hemianopsia, 486, 487
  - binasal, 489
  - bitemporal, 489
  - homonymous, 492
  - in brain-tumor, 537
  - in hysteria, rarely, 216, 492
  - in migraine, 611
  - location of lesion in, 490

Hemianopsia, method of determining, 489
  significance of, 490
  use of perimeter in determining, 490
Hemiatrophy, facial, 35
Hemicrania. *See* Migraine.
Hemidyschromatopsia, 493
  homonymous, 493
  in hysteria, 493
Hemihypertrophy, facial, 37
Hemilateral myelitis, table of symptoms of, 125
Hemin, test for, 594
Hemiopic pupillary inaction, 486
Hemiplegia, anatomy of brain-areas involved in, 136
  appearance of nails in, 59
    of tongue in, 154
  bedsores in, 202
  crossed, due to bulbar lesions, 149
  diagnosis of, 142, 143
  from acute infantile paralysis, 148
  from cerebellar hemorrhage, 146
  from cerebral hemorrhage, 143
      in crus cerebri, 145
      in frontal lobe, 145
      in island of Reil, 146
      in occipital lobe, 145
      in parietal lobe, 145
      in pons Varolii, 144
      location of lesion in, 143
    syphilis, 148
  from diffuse cerebral sclerosis of one hemisphere, 148
  from disseminated sclerosis, 148
  from hæmatoma of dura mater, 148
  gait in, 101
  hyperæsthesia in, 238
  in ingravescent apoplexy, 144
  in locomotor ataxia, 148
  in paretic dementia, 148
  in purulent meningitis, 149
  in renal disease with uræmia, 149
  irregular forms of, 146
  pupillary immobility in, 485
  spastic, 147
    due to cerebral tumor, 147
  symptoms associated with, 142, 143, 144
Hemoglobin affected by mercury in syphilis, 386
  greatly decreased in chlorosis, 385
      in leukæmia, 386
  of individual red blood-corpuscles greater than normal in pernicious anæmia, 384
Hemoglobinometer, Dare's, 379
  von Fleischl's, 376
Hemoglobinuria, accompanied by vomiting, 593
      urine in, 593
  causes of, 412
  signs of, 413
Hemoglobinuria, symptoms associated with, 533
Hemometer. *See* Hæmoglobinometer.
Hemophilia, 412
  hematemesis in, 587
  hematuria in, 412
  hemoptysis in, 601
  hemorrhage into retina in, 499
  joint-involvements in, 134
Hemoptysis, causes of, 312, 601
  diagnosis of, 587, 601
  significance of currant-jelly clots in, 601
Hemorrhage, anæmia in, 384
  cerebral, vomiting in, 577
  facies in, 35
  from mucous membrane in yellow fever, 521
  from nose in leukæmia, 388
  from stomach, 587
  in typhoid fever, 506
  indicated by anxious restlessness, 20
  into brain, causing hemiplegia, 143
        ocular paralysis, 475
      diplopia a symptom of, 466
  into membranes of cord, 223
  into retina, 500
  into skin, 184
  jaundice after severe prolonged, 176
  meningeal, symptoms of, 545
  movements minute, though active in, 20
  pallor of face in, 28
  pulmonary diseases in which it occurs, 601
  pulse of "empty arteries" in severe, 365
  subdural, cause of, 553
  ventricular, differential diagnosis between meningeal hemorrhage, 146
Hemorrhagic infarction of the intestine, 341, 584
  pleural effusion, causes of, 290
  pleurisy, 290
  smallpox, hæmoptysis in, 601
  retinitis, 499
Hemorrhoids, anæmia in bleeding, 384
  blood in stools caused by, 457, 459
  pruritus due to, 240
Henoch's disease, 183
Hepatic abscess, tongue in, 154
  cirrhosis, hæmatemesis in, 587
    with pigmentation, 174
  colic, 172, 619
    retraction of abdominal wall in, 322
Hepatitis. *See* Abscess, hepatic.
Hepatogenous jaundice, 170
Hereditary cerebellar ataxia, 96
  chorea, 78
  spastic paralysis, 105
  spinal ataxia, 96

Hernia, umbilical, 328, 347
Herpes labialis after salicylic acid, 199
  in croupous pneumonia, 31, 199
  in epidemic spinal meningitis, 199
 zoster, 200
Hiccough, 576
 causes of, 576
 significance of persistent, 576
Hip, spontaneous dislocation of, following infectious disease, 134
Hip-disease, character of pain in, 23, 615
Hippocratic face, 35, 451
 succussion, 279
Hippus, 486
Hodgkin's disease, differential diagnosis of, from true leukæmia, 388
  enlargement of cervical lymphatic glands in, 56, 519
  temperature in, 519
Hollow-chest as an indication of tuberculosis, 19
"Humming-top" murmur in chlorosis, 385
Hunger, character of child's cry in, 24
Huntingdon's chorea, 78, 573
Hutchinson pupil, in cerebral hemorrhage, 145
 teeth, 163
Hyaline casts in urine, 422
Hydatid cyst of liver, 353
 disease of spleen, 358
Hydrarthrosis, chronic, 130
Hydrocephaloid disease, 570
 state, 55
  Symes table showing differentiation of, from cerebral effusion, 55
Hydrocephalus, contraction of hands and arms in, 75
 face in, 30
 head in, 52
 nystagmus in, 482
 open fontanelle in, 53
 spastic rigidity of arms in, 75
Hydrochloric acid, test for, in stomach-contents, 338, 339
Hydronephrosis, 346
Hydropneumothorax, coin-percussion in, 288
 Hippocratic succussion in, 279
 metallic tinkling in, 275
 physical signs of, 288
Hyperæsthesia, 237
 following the use of drugs, 239
 in brain-tumor, 238
 in cerebro-spinal meningitis, 238
 in chronic alcoholism, 239
  leptomeningitis, 238
 in gout, 238
 in hemiplegia, 238
 in hysteria, 237
Hyperæsthesia in leprosy, 239
 in locomotor ataxia, 238
 in menopause, 238
 in myelitis, 238
 in neuralgia, 238
 in neurasthenia, 238
 in peripheral neuritis, 238
 in poisoning by lead and arsenic, 239
 in relapsing fever, 238
 in rickets, 239
 in scurvy, 239
 in transverse myelitis, 238
 in typhoid fever, at convalescence, 238
Hyperpyrexia, 501
 causes of, 504
Hypertrophy, cardiac, apex-beat in, 256
  associated with chronic contracted kidney, increase of urine in, 408
  diagnosis of, 314
  great, force of pulse in, 366
   in chronic interstitial nephritis, 447
  limited bulging of chest in, 246
  symptoms and signs of, 314
Hysteria, allochiria in, 236
 alterations of color-fields in, 493
 anæsthesia in, 216
 aphonia in, 631, 632
 bilateral anæsthesia in, 221
 borborygmi in, 111
 brachial monoplegia in, 85
 bromidrosis in, 205
 character of convulsions in, 565
  of cough in, 599
 contractions of hands, feet, and legs in, 74, 111
 diagnosis of, 564
 diarrhœa in, 457
 dilated pupil in, 465
 emprosthotonus in, 565
 facial spasm in, 50, 565, 566
 false clonus in, 630
 fatuous expression in, 32
 fever in, 529
 gait in paralysis of, 101
 gauntlet or stocking form of anæsthesia in, 219
 globus hystericus in, 566
 hemianæsthesia in, 216
 hemianopsia rare in, 216, 492
 hemidyschromatopsia in, 493
 hemorrhages into the skin in, 184
 hiccough in, 576
 hippus rare in, 486
 hysterogenous zones of hyperæsthesia in, 237
 localized sweating in, 205
 mirror-writing rare in, 82
 nodding spasm in, 50

- Hysteria, ocular symptoms of, 493
  - opisthotonus in, 565
  - pain, cutaneous in, 239
  - painful joints, 616
  - paraplegia in, 111
  - phantom tumor in, 347
  - ptosis in, 44
  - respirations in, 251
  - spasm of œsophagus in, 165
    - of tongue in, 162
  - spontaneous gangrene of skin in, 201
  - squint in, 481
  - status epilepticus in, 565
  - subnormal temperature in some cases of, 530
  - tremor of hands in, 81
  - urine increased in, 408
  - visual changes in, 216
  - vomiting in, 590
  - wry-neck in, 51
- Hysterical chorea, 78
  - mutism, 632
- Hystero-epilepsy, diagnosis of, 567
- Hysterogenous zones, 237

- ICHTHYOSIS of tongue, 158
  - Icterus. *See* Jaundice.
    - neonatorum, 177
    - urobilin, 176
- Idiocy, 52, 53
  - amaurotic family, 104
- Idiosyncrasy to drugs, 18, 184
  - hæmoglobinuria of, 412
- Impaction of feces, 343
- Impetigo contagiosa, appearance of eruption in, 198
    - symptoms of, 198
- Incontinence of urine, ammoniacal odor in, 18
    - due to concentrated urine, 405
    - due to excessive reflex irritation of bladder walls, 405
    - due to insensitive urethra, 405
    - due to loss of power of sphincter, 405
    - erythema of thighs in, 192
    - nocturnal, in children, 405
    - trousers stained in, 18
- Incubation-period of eruptive diseases, 189
- Indicanuria, 440
  - diseases in which it occurs, 415
  - in intestinal obstruction due to impaction, 585
  - test for, 415, 440
- Indigestion, anæmia in chronic, 384
  - character of respiration of child with, 24, 25
  - erythema in, 187
  - headache associated with, 532
  - hiccough in, 576
  - intestinal, pain in, 620
- Indigestion, muscæ volitantes in severe, 500
  - retraction of head in neurotic children with, 52
  - vertigo in, 547
- Infantile cerebral paralysis, gait in, 99
  - remittent fever, 520
  - spinal paralysis. *See* Anterior poliomyelitis.
- Infarction, renal, blood-casts in, 420
  - hæmaturia in, 409
  - hemorrhagic, of the intestine, 341, 584
- Influenza, epidemic, cardiac failure in, 525
    - catarrhal symptoms of, 525
    - character of fever in, 525
      - of sputum in, 602
    - complications of, 525
    - diarrhœa, 525
    - differential diagnosis from dengue, 521
    - great prostration in, 525
    - hæmatemesis in, 587
    - hyperæsthesia in, 239
    - micro-organism of, as cause of empyema, 290
    - ocular palsy in, 476
    - pain in back in early stages of, 624
    - vomiting in, 525
- Ingravescent apoplexy, 144
- Injury, expression of anxiety after severe, 29
  - iritis due to, 499
  - spinal, knee-jerk decreased immediately after, 627
- Insomnia in Malta fever, 512
- Inspection of chest, 241
  - of abdomen, 321
- Insufficiency of convergence in exophthalmic goitre, 465
- Intellectual face, 27
- Intention-tremor of cerebral palsy of children, 74
- Interlobular pleurisy, physical signs of, 291
- Internal squint, causes of, 477
- Intestinal indigestion, pain in, 620
  - obstruction, 343
    - albumin in urine, 583
    - constipation in, 450
    - due to cancer or stricture, 584
    - due to impaction of foreign body, indicanuria in, 584
      - symptoms of, 584
      - vomiting in, 584
    - due to intussusception, blood-stained mucous evacuations of children in, 580
      - detection of tumor in, 581
      - distention of abdomen in, 581

Intestinal obstruction due to inequality of shape of abdomen in, 581
    pain, character of, in, 579
    patulous condition of anus in, 581
    symptoms of chronic, 581
    tenesmus in, 581
    vomiting early in, in invagination, 579
        due to completeness of obstruction, 579
  due to strangulation by bands, 582
    character of constipation in, 582
      of localized tenderness and percussion-dulness in, 583
      of pain in, 582, 620
      of pulse in, 582
        symptoms of, 341, 582
        tympanitic distention in, 583
      of vomiting in, 582
  due to strangulation by Meckel's diverticulum a cause of, 583
    pain in, 582
    temperature in, 582
    tympanitic distention in, 583
    urine diminished in, 583
  due to volvulus, vomiting not a constant symptom of, 584
  hiccough in, 576
  indicanuria in, 415
  leucocytosis in, 389
parasites, 460
perforation, pain in, 618
rigidity, 347
Intestine, hemorrhagic infarction of the, 584
Intraocular muscles, paralysis of, 482
Intussusception, sloughing of bowel in, 460
Iritis due to injury, 499
  due to rheumatism, 499
  due to syphilis, 499
  headache due to, 534
  immobile pupil in, 465
Irritative myosis, 485
Itching of jaundice, 174

JACKSONIAN epilepsy, 557
Jaundice, 170
  as result of gastroptosis, 174
  bradycardia in, 317
  catarrhal, 171
  cause of, 175
    complicating diabetes, 174
Jaundice, cause of constipation occurring in, 449
    of hæmatogenous, 175, 176
    of hepatogenous, 170, 171
  Charcot's fever in, 175
  color of urine in, 175, 414
  diabète bronzé in, 174
  fatty diarrhœa in, 457
  from cerebral concussion, 177
  from fright or extreme anger, 177
  hæmatogenous, 175
  headache in, 533
  hepatogenous, 170
  in acute phosphorus-poisoning, 173, 591
    ulcerative endocarditis, 177
    yellow atrophy, 173, 175
  in amyloid liver, 174
  in chronic valvular heart disease, 173
  in cirrhosis of liver, 173
  in croupous pneumonia, 176
  in diabetes, 174
  in disease of the pancreas, 174
  in echinococcus of liver, 174
  in malignant disease, 173
  in new-born, 177
  in pressure from aneurism, 173
  in remittent malarial fever, 520
  in Weil's disease, 173, 520
  in yellow fever, 173, 521
  itching of, 174
  method of examining for, 170
  obstructive, 172
  peptonuria in, 435
  persistent and progressive in carcinoma of pancreas, 341
  pruritus in, 174, 239
  purpuric eruption in severe, 184
  pyæmic, 176
  stools in, 175, 176, 459
  sweating in, 205
  symptoms of catarrhal, 171
  tongue in, 153
  symptoms of hepatogenous, 171
  urine in, 175, 414
  vomiting in, 592
Joints, the, 129
  alterations of, in acute articular rheumatism, 130
      synovitis, 130
    in locomotor ataxia, 130
  different modes of infection of, in gonorrhœal arthritis, 130
  enlargement of, in rheumatoid arthritis, 131
  hypertrophic osteo-arthritis following typhoid fever, 132, 133
  in acute epiphysitis of infancy, 132
    synovitis, 130
  in central myelitis, 132
  in cerebro-spinal meningitis, 132
  in chronic lead-poisoning, 132

- **Joints in dengue, 133**
  - in epidemic dysentery, 133
  - in gout, 132
  - in hæmophilia, 134
  - in milk-leg, 134
  - in osteomyelitis, 132
  - in rheumatoid arthritis, 131
    - septic, 132
  - in scarlet fever, 133
  - in Schönlein's disease, 133
  - involvement of, with fever, in osteomyelitis, 528
    - in purpura, 528
    - in pyæmia, 528
  - small, affected in Morvan's disease, 129
    - in syringomyelia, 129
  - violent pain on motion of, in Malta fever, 512
- **Jumpers, the, 78.** *See* Saltatoric spasm.

- **KERNIG'S SIGN in meningitis, 542**
- **Kidney, amyloid disease of, 447**
  - blood-casts in acute inflammation of, 420
  - cancer of, hæmaturia due to, 410
  - character of blood in urine from, 409, 411
  - chronic contracted, accentuation of heart-sounds in, 298
  - cystic degeneration of, 346
  - fatty casts in fatty degeneration of, 422
  - floating, 328, 343
  - hæmaturia due to stone in, 411
  - hydronephrosis, 346
  - irritation of, cylindroids found in, 422
  - methylene-blue test to determine activity of, 418
  - multiple abscess of, pus-casts in urine in, 420
  - pain in, 619
  - septic infection of, casts of micrococci in, 421
    - hæmaturia due to, 409
  - slow degeneration of parenchyma of, granular casts in, 421
  - tuberculosis of, 411, 621
- **Klumpke's paralysis, 88**
- **Knee-jerk, diseases in which it is decreased, 627**
    - in which it is increased, 628
  - how best elicited, 625
  - reinforced, 625
- **Kyphosis, 38**

- **LA GRIPPE.** *See* Influenza.
- **Lactic acid, test for, in stomach-contents, 338**
- **Landouzy-Déjérine type of muscular atrophy, 33**
- **Landry's paralysis, diagnosis of, 114, 529**
    - loss of knee-jerk in, 627
    - loss of reflexes, predominant symptom of, 529
    - non-spastic paraplegia in, 114
- **Laryngeal crisis in locomotor ataxia, 92**
  - obstruction, cyanosis due to, 181
  - paralysis, table showing causes of, 632
  - phthisis, cough in, 598
  - spasm, 294
- **Laryngismus stridulus in tetany, 571**
- **Laryngitis, night-cough in, 599**
  - partial or complete loss of voice in, 598
  - short, sharp, brassy cough in, 598
  - whispering voice in, 631
- **Laveran, malarial germ of, 389**
- **Lead colic, retraction of abdominal wall in, 322**
  - -poisoning, chronic, paralyses in, 76
    - peripheral neuritis in, 230
    - tremors in, 80
- **Leprosy, appearance of hands in, 67**
  - gangrene in, 67
  - hyperæsthesia in, 239
  - leonine face of, 34
- **Leptomeningitis, diagnosis of coma in purulent, 554**
  - hyperæsthesia in chronic, 238
- **Leucocytes, 367**
  - mononuclear, 381
  - polynuclear, 382
  - transitional, 381
- **Leucocythæmia.** *See* Leukæmia.
- **Leucocytosis, 388**
  - in appendicitis, 389
  - in cholangitis, 519
  - in intestinal obstruction, 389
    - perforation during typhoid fever, 389, 509
  - in pneumonia and other infectious diseases, 388
  - in trichinosis, 388
  - terminal, 389
- **Leucoderma in goitre, 179**
- **Leucokeratosis buccalis, 158**
- **Leucoma of tongue, 158**
- **Leukæmia, hæmaturia in, 412**
  - lymphatic, enlargement of superficial lymph-glands in, 388
    - lymphocytes alone increased in, 382, 388
  - spleno-medullary, blood in, 386
    - enlargement of spleen in, 356, 388
    - symptoms of, 388
  - uric acid in urine in, 425

Lichen planus, appearance of tongue in, 158
  ruber, appearance of nails in, 59
Lienteric diarrhœa, 454
Lineæ albicantes, 321
Lingual paralysis, 160
  ulcers, 156
Lips, blueness of, in heart disease, 312
  fulness of, in persons of strong sexual appetite, 28
    indicating phlegmatic temperament, 28
  greatly thickened in cretinism, 34
  immobility of, due to mucous patches or ulceration of buccal mucous membrane, 31
  pendulous, in progressive bulbar paralysis, 47
  slightly parted, dry, and cyanotic in chronic pulmonary or cardiac disease, 31
  thin and mobile, in nervous individuals, 28
  twitching of raised upper, in peritonitis or pain below diaphragm, 30
Lithæmia, cylindroids in urine in, 423
Litten's sign, 253
Liver, acute yellow atrophy of. *See* Yellow atrophy, acute.
  amyloid disease of, jaundice in, 174
  causes of tenderness of, 352
  cirrhosis of, jaundice in, 173
  enlarged, position in, 21
    and smooth, in amyloid disease, 349
      of kidney, 447
  enlargement of, 328
    position in, 21
  floating, 352
  hæmatemesis in, 587
  headache in congestion of, 532
  hydatid cyst of, 353
  indicanuria in cancer of, 415
  jaundice in, 173, 175
  malignant disease of, jaundice in, 173
  nodules and umbilication of, in malignant disease of, 349
  percussion-note over, 348
  pushed down by right-sided pleural effusion, 349, 352
  rough, in cirrhosis, 349
  swelling of, in Weil's disease, 520
  symptoms of cirrhosis of, 349, 351
    of malignant disease of, 349
    of tropical abscess of, 352
      vomiting in, 593
Localization of functions of segments of spinal cord, 117
Localized sweating, 205
Locomotor ataxia, 91
    allochiria in, 236
Locomotor ataxia, alteration of joints in, 130
  anæsthesia of lower portion of body and of legs in, 221
  analgesia in, 221
  Argyll-Robertson pupil in, 92, 484
  athetoid movements in advanced, 82
  bilateral atrophy of tongue in, 159
  bladder-symptoms of, 404
  blunted and delayed sensation in, 221
  brachial monoplegia in, 87
  cardiac feebleness in, 318
  coffee-ground vomit in gastric crisis of, 586
  contracted pupil in, 485
  cutaneous hemorrhages in, 184
  diagnosis of, 627
  diarrhœa as an intestinal crisis of, 453
  differential points between Friedreich's ataxia and, 92
  diplopia in, 45, 466
  double sciatic pain in, 616
  flat-foot in, 129
  gait of, 91, 94
    cause of, 92, 93
  gastric crisis of, 92, 586
  girdle-sensation in 236, 237
  hæmaturia in, 412
  hemiplegia in, 148
  hyperæsthesia in, 238
  inability to use fingers and hands in, 82
  laryngeal crisis of, 92
    spasm in, 294
  lesion of optic nerve in, 497
  loss of knee-jerk in, 627
  muscular atrophy of arm in, 87
    hypotonus in, 92
  numbness of feet in, 92
  nystagmus in advanced, 482
  ocular symptoms of, 497
  optic atrophy in, 497
  pain in, 509, 613, 614, 616
    of skin in, 239
  paræsthesia in, 235
  paraplegia in, 114, 122
  perforating ulcer in, 134, 201
  ptosis in, 45, 466
  retention of urine in, 404
  Romberg's symptom of, 92
  swaying of body with eyes closed in, 92
  syphilis a frequent cause of, 93
  table of symptoms of, 92, 93
  thoracic pain in, 613
  twitching of fingers in, 82

Locomotor ataxia, vesical crisis of, 92
    Westphal's sign of, 92
Löffler's methylene-blue mixture, 396
Lordosis in cretinism, 34
  in progressive muscular atrophy, 70
Lumbago, pain in, 615, 624
Lumbar puncture, 522
    in diagnosis of cerebro-spinal meningitis, 522
Lupus, ulceration of tongue in, 157
Lymphangioma, congenital, tongue in, 159
Lymphatic leukæmia, 388
Lymphocytes, 381
Lyons's method of estimating urea, 440
Lysis, fever in erysipelas, ending by crisis or, 515
    in scarlet fever, ending by, 513
    in smallpox, ending by, 515
    in typhoid, ending by, 506
  in catarrhal pneumonia, 526
  rare in croupous pneumonia, 526

MACROGLOSSIA, 159
  Macula lutea, 496
Maddox-rod test, 468
Madura foot, 68, 135
Mainen griffe, 69
Malaria, anæmia in, 385
  aortitis rare in, 613
  blood in, 389
  brow-ague in, 611
  Chenzynski solution for staining organisms of, 396
  Cheyne-Stokes breathing in hæmaturic, 252
  chill in, 501
  coma in, 552
  convulsions due to, 563
  enlargement of spleen in, 358
  Futcher's method of staining organisms of, 397
  hæmatemesis rare in, 587
  hæmaturia in, 409, 410
  hæmoglobinuria in, 412
  headache in, 546
  intermittent, attacks occurring earlier each day in, 517
    characteristic of fever, quartan, 516
      quotidian, 516
      tertian, 516
    chill, fever, and sweats in, 515
      diagnosis of, 516
      effect of quinine on, 517
  method of examining organism of, 396
    of staining organism of, 393, 396
  muddy yellow skin in chronic, 178
  parasite of æstivo-autumnal, 392
    of quartan, 391, 394
    of tertian, 390, 394
Malaria, pernicious, differential diagnosis between yellow fever and, 521
  remittent, bilious vomiting in, 520
    character of fever in, 520
    diagnosis between typhoid fever and, 510
      between yellow fever and, 521
    due to æstivo-autumnal parasite, 520
    jaundice in, 520
    sweating in, 204
    synonyms of, 520
  retinal hemorrhage in, 499
  roseola in, 189
  splenic enlargement in, 356
Malarial germ of Laveran, 389
Malignant pancreatitis, 340
  pustule. *See* Anthrax, malignant.
Malingering, convulsions in, 569
  hæmatemesis in, 587
  inability to ape facies of disease in, 29
  merycismus in, 594
  sciatica in, 616
Malta fever, anorexia in, 512
    character of fever in, 512
    frequency of relapse in, 512
    headache in, 512
    insomnia in, 512
    pain on motion in, 512
Mammary gland, enlargement of, in pulmonary tuberculosis, 249
Maniacal chorea, 77
Marasmus, sinking in of fontanelle in, 54
Marchand's solution, 397
McBurney's point, 617
Measles, character of fever in, 514
  date of eruption in, 188
  diazo-reaction in urine of severe, 445
  headache in, 545
  persistence of fever in, due to bronchial or gastro-duodenal catarrh, 514
  rash of, 188
    on pharynx and buccal mucous membrane, 167
  symptoms of, 188
Meckel's diverticulum, 583
Mediastinal abscess, diagnosis of, from mediastinal tumors, 296
  growths, as causes of laryngeal spasm, 294
    bulging of chest in, 247
    caput Medusæ in some cases of, 327
    diagnosis of, from abscess, 296
      from aneurism, 295
      from chronic pneumonia, 295
      from pericarditis, 297
      from pleural effusion, 296

- Mediastinal growths, dysphagia in, 162
    - hoarseness of voice due to pressure by, 631
    - laryngeal cough due to pressure on larynx by, 598
    - pain in, 614
    - signs and symptoms of, 295
- Mediastino-pericarditis, indurative, pulsus paradoxus in, 363
- Megaloblasts, called corpuscles of Ehrlich, 384
  - in pernicious anæmia, 384
- Megalocytes, 368
- Megrim. *See* Migraine.
- Melæna neonatorum, causes of, 587
    - vomiting in, 587
- Melancholia, facial expression in, 32
  - puerperal lingual spasm in, 162
  - with oxaluria, diagnosis of, 425
- Melanotic cancer, black urine in, 409
- Ménière's disease, symptoms of, 547
    - vomiting following tinnitus aurium and vertigo in, 592
- Meningeal tuberculosis, Cheyne-Stokes respiration in, 252
- Meningitis, basilar, lesions causing ocular paralysis in, 475
    - papillitis in, 497
    - retraction of head in, 51
  - bilateral anæsthesia in, 221
    - loss of knee-jerk, 627
  - bulging fontanelle in purulent, 53
  - cerebro-spinal. *See* Cerebro-spinal meningitis.
  - contracted pupil in, 465
  - contraction of hand in, 75
  - diplopia in, 466
  - facial spasm in, 48, 49
  - hippus in early stages of acute, 486
  - hyperæsthesia in cerebro-spinal, 238
  - Kernig's sign, 542
  - lumbar puncture, 522
  - nystagmus occasionally in, 482
  - occipital headache in, 578
  - pain in nape of neck in, 578
  - peptonuria not present in tubercular, 435
  - piercing cry in, 24
  - pseudo-, 570
  - purulent, hemiplegia in, 149
    - vomiting in, 578
  - reflexes in, 627
  - rigidity of dorsal muscles in, 578
  - scaphoid belly in tubercular, 322
  - spinal, loss of knee-jerk in, 627
  - subnormal temperature in some cases of, 530
  - symptoms of, 540
  - *tache cérébrale* in, 185
  - tubercular, 540
  - vomiting in, 578
- Menstruation, anæmia due to profuse, 384
  - dark areas under eyes during, 28
  - disorders of, in chlorosis, 385
  - hæmatemesis as vicarious, 587
  - hæmoptysis as vicarious, 601
- Merycismus, 594
- Mesenteric glands, tuberculosis of, 340
- Mesentery, cysts of, 346
- Metallic tinkling heard over chest, in cavity, 275
      - over hydropneumothorax, 275, 288
      - over stomach, 275
- Metatarsal neuralgia, 624
- Methylene-blue test to determine activity of kidneys, 418
- Microcephalus, 54
  - head in, 52
- Microcytes, 368
- Micro-organism of influenza as a cause of empyema, 290
- Micturition, frequent, in renal disease, 621
- Migraine, hæmianopsia in, 532
  - hyperæsthesia in, 238
  - muscæ volitantes in, 499
  - scotomata in, 593
  - sweating of head in, 205
  - symptoms of, 611
  - vomiting in, 593
- Miliaria, 205
- Milk-leg, joints in, 134
- Milky-looking urine, 415
- Mirror-writing, 82
- Miscarriage, a symptom of syphilis, 22
- Mitral disease, 299
  - obstruction, 300
    - and regurgitation, accentuation of pulmonary second heart-sound in, 298, 301
  - regurgitation, 300
    - præcordial thrill in, 302
    - pulse in, 363
    - symptoms of, 312
  - stenosis, pulse in, 363
    - reduplication of heart-sounds in, 299
    - thrills in, 258
- Moebius's sign, 465
- Möller-Barlow's disease, 132
- Mono-anæsthesia, 218
- Mononuclear leucocytes, 381
- Monoplegia, 84, 125
  - brachial, 84
    - bilateral, 88
  - of lower extremities, 125
  - spastic, 108
- Morbilli. *See* Measles.
- Morphœa as a cause of facial hæmiatrophy, 37
- Morton's painful toe, 624

- Morvan's disease, 72
    - gangrene of hands in, 68
    - hand similar to claw-hand in, 72
    - small joints affected in, 129
- Mosquito as a carrier of malarial parasites, 394, 395
- Mouth, chapter on, 150
    - constant opening of, in heart disease, 31
    - dryness of, in paralysis of salivary glands, 39
    - pigmentation of, 318
- Mouth-breathers, 31
    - nasal twang of voice in, 632
- Movements of tongue, 160
- Mowing gait, 101
- Mucous disease, appearance of tongue in, 153
    - patches about mouth and anus in infantile syphilis, 30
        - immobility of lips due to, 30
- Multiple sclerosis. *See* Sclerosis, disseminated.
- Mumps, enlarged parotid glands in, 56
    - peptonuria in, 435
- Murmur, aortic regurgitant, 304
    - capillary pulsation in, 313
    - Corrigan's pulse in, 305, 313
    - dyspnœa in, 312, 313
    - ox-heart in, 305
    - pulsation of retinal arteries in, 313
    - pulse-wave of, 363
    - Quincke's sign of, 313
    - short and sharp pulse in, 363
    - symptoms of, due to failing compensation, 312
    - water-hammer pulse in, 305, 313
    - stenosis, 302
        - cardio-pulmonary, 299
        - small and hard pulse in, 363
        - symptoms of, due to failing compensation, 312
    - hæmic, over fontanelle in rickets, 55
    - humming-top, in jugular vein in chlorosis, 385
    - mitral regurgitation, 300
        - small volume of pulse in, 363
        - symptoms of, due to failing compensation, 312
        - thrill in, 302
    - stenosis, 302
        - gallop-rhythm in, 303
        - pulse-wave of, 365
        - small volume of pulse in, 363
- Murmur, mitral stenosis, symptoms of, due to failing compensation, 303, 312
        - thrill in, 303
    - pulmonary regurgitation, 309
        - stenosis, 309
    - splenic, in leukæmia, 388
    - tricuspid regurgitation, 309
        - stenosis, 309
        - systolic, in emphysema, 292
- Murmurs, relative frequency of associated, 310
    - gravity of, 311
    - venous, in anæmia, 384
- Muscæ volitantes, 499
- Muscle-tone, 625
- Muscles, contraction of, in paramyoclonus multiplex, 78
        - in electric chorea of Bergeron, 78
        - in Huntingdon's chorea, 78
        - in "jumpers," 78
        - intraocular, 482
        - shock-like, in Dubini's disease, 78
    - ocular, paralysis of, 467
- Muscular atrophy, excessive sweating of hands in, 60
    - facio-humero-scapular type of, 33
    - idiopathic. *See* Atrophy, idiopathic muscular.
    - progressive, claw-hands in, 70
- Mutism, hysterical, 632
- Myalgia, pain in, 615
- Mycetoma, 68, 135
    - swelling of hand, followed by rupture in, 68
- Mycoses, appearance of tongue due to growth of, 153
- Myelitis, acute ascending, bedsores in, 202
    - character of fever in, 529
    - symptoms of, 115, 529
    - transverse, allochiria in, 236
        - anæsthesia in, 222
        - bilateral anæsthesia in, 222
        - bladder-symptoms of, 403
        - collateral symptoms of, 405
        - diagnosis of cervical, dorsal, and lumbar, 120
        - effect of lesion in, 116
        - girdle-sensation in, 106, 237
        - hyperæsthesia in, 238
        - knee-jerk in, 627, 628
        - location of lesion in, 119
        - non-spastic paraplegia in, 115
        - paræsthesia in, 236
        - sensory paralysis in, 106
        - spastic paraplegia in, 106
    - chronic, gait of, 97

- Myelitis, hemilateral, symptoms of, 125
  - hiccough in, 576
  - subacute, symptoms of, 116
  - traumatic, retention of urine in, 403
- Myelocyte of Ehrlich in spleno-medullary leukæmia, 387
- Myelopathic albumosuria, 434
- Myocarditis, degenerative, 318
- Myoclonus multiplex, 78
- Myosis in facial hemiatrophy, 37
  - irritative, 485
  - paralytic, 485
- Myotonia congenita, 79, 575
- Myxœdema, diagnosis between dropsy and, 206
  - enlargement of feet in, 128
    - of tongue in, 160
  - face of, 34, 38
  - skin in, 180
  - spade-like hand in, 68
  - symptoms of, 210

- NAILS, appearance of, in acute fevers, 59
      - infantile palsy, 59
    - in anæmia, 59
    - in chloral-habit, 60
    - in eczema, 59
    - in gout, 59
    - in hemiplegia, 59
    - in lichen ruber, 59
    - in prolonged illness, 59
    - in psoriasis, 59
    - in pulmonary osteo-arthropathy, 59
    - in Raynaud's disease, 59
    - in sclerodactyle, 59
    - in syphilis, 59
    - in syringomyelia, injury, or neuritis, 59
  - atrophy of, 59
  - hypertrophy of, 59
  - white spots on, 59
- Nasal catarrh, chronic atrophic, breath in, 19
  - obstruction, facial expression in, 30
- Nausea, acute, position in, 20
- Neck, spasm of the muscles of the, 50
- Necrosis, anæmic, 318
  - fat, from impacted gall-stone, 341
- Nephritis. *See* also Kidneys and Bright's disease.
  - acute, diarrhœa in, 453
    - diffuse, anæmia in, 446
      - anasarca in, in children, 446
      - heart slightly dilated in, 447
      - high arterial tension in, 446
      - nausea and chilly sensations preceding, 445
      - prognosis of, 446
- Nephritis, acute diffuse, puffiness of face about eyes in, 446
      - pulse hard and tense in, 446
      - skin harsh and dry in, 446
      - symptoms and signs of, 445, 446
      - urine in, 445
    - hemorrhagic effusions into pleura in, 290
    - irritation of bladder in, 405
    - parenchymatous, decrease of urine in, 408
      - epithelial casts in urine a positive sign of, 420
      - fever in, 527
      - symptoms of, 527
  - chronic hemorrhagic, hæmaturia in, 410
    - interstitial, albumin transient in, 447
      - atheroma of blood-vessels in, 447
      - chronic bronchitis in, 447
      - diagnosis between brain-tumor and, 537
      - dropsy rare in, 447
      - excess of uric acid in urine often antedates, 425
      - frequency of urination in, 446
      - headache in, 535
      - heart markedly hypertrophied in, 447
      - high tension of pulse in, 362, 447
      - hyaline casts in urine of, 422
      - low specific gravity of urine in, 408, 447
      - nocturnal urination in, 446
      - pruritus in, 239
      - pulmonary œdema in, 447
      - shortness of breath most marked at night in, 447
    - parenchymatous, decrease of urine in, 408
      - heart somewhat hypertrophied in, 447
      - marked tendency to anasarca in, 446
      - prognosis in, 446
      - uræmic vomiting and coma may occur in, 446
- Nervous bladder, 406
  - diarrhœa, acute, 453
- Neuralgia, causes of, 610
  - due to lead- or arsenic-poisoning, 611
  - due to malaria, 611
  - hyperæsthesia in, 238
  - intercostal, causes of, 613, 614
    - pain in, 614
    - spots of Valleix in, 613

- Neuralgia, Morton's painful toe in, 624
  - occipital, causes of, 612
    - diagnosis of, 612
    - pain in, 612
  - œdema in, 209
  - of fifth nerve, infra-orbital, symptoms of, 611
      - third division, symptoms of, 611
      - upper branch, symptoms of, 611
  - of foot, 624
  - of head, due to decayed teeth, 610
    - due to diseased ear, 610
    - due to overstrained eyes, 610
    - in anæmic, overworked women, 610
  - of labia majora or perineum, 614
  - of pelvic viscera in women, 614
    - pain in, 610
  - sciatic, pain in, 615
  - supraorbital, diagnosis of, from neuritis, 611
- Neurasthenia, gait in, 18
  - headache in, 534
  - hyperæsthesia in, 238
  - increase in urine in, 408
  - knee-jerk increased in, 628, 629
  - paræsthesia in, 236
  - scapulo-humeral reflex increased in, 626
  - vomiting in, 590
- Neuritis, acute multiple, character of fever in, 529
      - knee-jerk lost in, 627
      - symptoms and signs of, 529
  - anæsthesia in toxic peripheral, 230
    - in 50 per cent. of diphtheritic, 231
  - as a cause of anæsthesia, 228
  - claw-hand in peripheral, 70
  - diagnosis of, 627
  - diphtheritic, 231
  - general anasarca in multiple peripheral, 207
  - hyperæsthesia in peripheral, 238
  - in foot, leg, and thigh, distribution of anæsthesia in, 234
  - in hand, distribution of anæsthesia in, 233
  - multiple, 529
  - optic. *See* Papillitis.
  - retro-bulbar, 497
  - sciatic, diagnosis of, 615, 616
    - heel and toe tender in, 616
    - knee-jerk in, 628
    - limping gait of, 90
    - pain in, 615
    - symptoms of, 615
    - tender joints in, 615
    - toxic peripheral, 230
- Neuroma, character of pain in, 624
- Neutrophile corpuscles, 382, 387
- New-born, cyanosis in, 180, 181
  - jaundice in, 177
- Nictitating spasm, 49
- Nocht's modification of Romanovsky's stain for malarial parasites, 393
- Nodding spasm, 50
- Noma, 165
- Normoblasts, 368
- Nose, anæmia due to hemorrhage from, 384
  - asthma due to reflex irritation in, 294
  - broad and flat, in cretinism, 34
  - broadness of bridge of, an indication of congenital syphilis, 30
  - dilated, in heart disease, 31
  - hemorrhage from, in leukæmia, 388
  - obstruction of, in mouth-breathers, 31
  - pinched and drawn, indicating pain in chest in children, 30
- Nystagmus, 481
  - in acute meningitis, 482, 579
  - in children otherwise normal, 482
    - with imperfect vision, 482
  - in disseminated sclerosis, 482
  - in epilepsy, 482
  - in Friedreich's ataxia, 482
  - in growths in cerebellum or pons, 482
  - in hydrocephalus, 482
  - in locomotor ataxia, advanced, 482
  - in multiple sclerosis, 482
  - in spastic paraplegia, 104

- OBERMEIER, spirillum of, 513
- Obstruction, intestinal. *See* Intestinal obstruction.
    - of the rectum, 585
      - symptoms of, 585
  - of air-passages, 294
  - of bile-ducts, causes of, 353, 354
  - of gall-bladder, symptoms of, 353
- Obstructive jaundice, 172
- Occupation-spasm, 76
- Ocular muscles, paralysis of, 467
- Oculo-facial paralysis, 47
- Œdema. *See also* Dropsy and general anasarca.
  - localized, 207
  - neonatorum, 210
  - of the retina in general arterial disease, 499
  - of tongue, 159
  - pulmonary, absence of fever in, 286
    - character of sputum in, 286
    - crepitant and bubbling râles in, 275
    - due to acute disease of lungs, 286
    - due to injury of vagus, 286

Œdema, pulmonary, dulness on percussion in, 286
        feeble, hesitating speech in severe, 632
        in chronic interstitial nephritis, 447
        liquid watery sputum in, 602
        sputum in, 286
Œsophagus, cancer of, 165
    diverticulum of, 165
    hysterical spasms of, 165
    spasmodic contraction of, 165
    stricture of, 164
        causes of, 165
        diagnosis of, 164
        dysphagia due to, 164, 165
Oligochromæmia, 368
Oligocythæmia, 367
Omentum, tuberculosis of, 340
    tumors of, 343
Onychogyrophosis, 59
Ophthalmoplegia, 43
Ophthalmoscopy, 493
Opisthotonos in hysteria, 565
    in severe meningitis, 578
Opium-poisoning, 549
    differential diagnosis between poisoning from alcohol and, 550
Oppenheimer'a modification of Dennigé's test for acetone in urine, 439
Optic atrophy, 497
    nerve, 493
    neuritis, 496
Orthophoria, 468
Osteitis deformans, head in, 53
        of cranial bones, headache in, 546
        triangular face of, 38
Osteomalacia, gait of, 102
Osteomyelitis, acute, arthritis in, 132
        joint-involvement with fever in, 528
Osteophytes, formation of, in rheumatoid arthritis, 131
Otitis in scarlet fever, 513
    media, connected with thrombosis of cavernous sinus, 544
        diagnosis between cerebro-spinal meningitis and, 523
    vertigo in, 547
Ovarian cysts, 324
Oxaluria accompanying spermatorrhœa, 426
    concomitant symptom of melancholia, 425
    cylindroids in urine of, 424
    due to ingestion of pears, cabbage, and tomatoes, 426
    yellow color of skin in, 177
"Ox-heart" of aortic regurgitation, 305
Oxyuris vermicularis, 461, 462
Ozæna, breath in, 19

PACHYMENINGITIS interna hæmorrhagica, coma in, 553
        headache in, 545
    spinal, spastic paraplegia in, 107
Pain below diaphragm, twitching of upper lip in, 32
    best manner of discovering true seat of, 22
    bilateral, of rheumatism of scalp, 612
    burning, in œsophagus, stomach, and abdomen in phosphorus-poisoning, 591
    causes of circumscribed abdominal, 622
        in back in children, 615
        in neighborhood of heart, 613
        in sacral region, 615
        neuralgic, 610, 613
        sciatic, 615
        supraorbital neuralgic, 611
    character of, 609
    colicky, extending to right shoulder in acute pancreatitis, 591
        in embolism of superior mesenteric artery, 591
    concomitant symptoms of abdominal, 24
    crisis of, in tabes, 92
    darting, in urethra, in cystitis, 404
    distribution of, in neuralgia of fifth nerve, 611
    expression about eyes in children due to, 30
        of face due to, 30
    in abdominal colic, 616
    in acute aortitis, 613
        bellyache, 20
        cholecystitis, 619
        pleurisy, 286, 614
    in aneurism, 307, 614, 621
    in angina pectoris, 613
    in appendicitis, 617
    in back, causes of, 624
    in cerebral tumor or abscess, 612
    in chest in aortic aneurism, 614
    in coxalgia, 23, 615
    in cystitis, 623
    in dengue, 624
    in diabetes mellitus, 622
    in diarrhœa, 23
    in diffuse hepatitis, 620
    in dysmenorrhœa, 622
    in entero-colitis, 453
    in fecal impaction, 620
    in fissure of the anus, 623
    in floating kidney, 619
    in gallstone colic, 619
    in gastralgia, 622
    in gastric cancer and ulcer, 332, 334, 614
    in hæmoglobinuria, 593
    in head in children, 29

Pain in hemorrhagic infarction, 584
in hepatic colic, 619
in hepatitis, 620
in intercostal neuralgia, 613, 614
in intestinal indigestion, 620
obstruction, 579, 580, 620
perforation, 341, 618
in intussusception, 597
in jaundice, due to gallstones, 172
in lead-colic, 620
in locomotor ataxia, 613, 616, 622
in lumbar region, due to perinephritic disease, 448
due to pyelitis, 448
due to stone in kidney or ureter, 448
in mediastinal growths, 614
in meningeal hemorrhage, 545
in migraine, 611
in neuralgia of foot, 624
of head, 610
of pelvic viscera, 614
in neuritis, 611
in neuromata, 624
in occipital neuralgia, 612
in one side of head in migraine, 611
in pancreatic calculus, 620
in pancreatitis, 618
in pericarditis, 614
in peritonitis, 618
in pseudo-angina pectoris, 613
in pulmonary diseases in children, 23
in pyelitis, 448
in renal calculus, 616
colic, 621
in rheumatism of scalp, 612
in rickets, 615
in sciatic neuralgia, 615
in scurvy, 615
in skin in tabes dorsalis or hysteria, 239
in spinal caries, 615
in syphilitic periostitis of skull, 612
in typhoid fever, 619
often referred to point distant from its source, 610
significance of, 609
signs of, 610
summary of conditions producing acute, sudden, 623
tearing, burning, in hemorrhage into membranes of cord, 223
thoracic, in locomotar ataxia, 614
throbbing, in inflammation, 609, 624
varieties of, 609
Pallor of face in advanced stage of acute alcohol-poisoning, 548
in chlorosis, 28, 180, 385
in fright, 28
in hemorrhage, 28
of skin in absence of pigment, 180
Pallor of skin in anæmia, 384
in vasomotor disturbance, 180
in vitiligo, 180
Palmus, 78
Palpation of chest, 255
Palsy, cerebral, of children, 73
increased knee-jerk in, 628
crutch-, 76
Pancreas, abscess of, 342
carcinoma of, 334
cysts of, 341
fatty diarrhœa due to disease of, 457
hemorrhagic inflammation of, 341
inflammation of, 341
pain in, 618
symptoms of, 591, 618
vomiting in, 591
jaundice in disease of, 174
Pancreatic calculus, pain in, 620
diabetes, 334
Pancreatitis, acute hemorrhagic, symptoms of, 341
malignant, 340
Papillitis, 496
in acute febrile disorders, 497
in anæmia, 497
in brain-tumor, 496, 536, 578
in cerebral abscess, 497
in meningitis, 497
tubercular, 497
in rheumatism, 497
in syphilis, 497
in toxæmia from alcohol, 497
from lead, 497
Papilloma of bladder, hæmaturia due to, 411
Paræsthesia, 235. *See* also Allochiria.
brachial, 89
from exposure to carbolic acid, 236
in aconite-poisoning, 236
in brain tumor, 236
in carbolic-acid poisoning, 236
in locomotor ataxia, 235
in myelitis, 236
in neurasthenia, 236
in spinal syphilis, 236
Paralysis agitans, festination in, 573
gait in, 100
Parkinsonian visage in, 32, 573
speech and voice in, 634
tremor of hands in, 79
wasting, rigidity, and flexion of hands in, 73
asthenic bulbar, 47
diagnosis between progressive bulbar paralysis and, 47
speech of, 634
bilateral facial, 46
in acute bulbar paralysis, 46
in bilateral inflammation at stylo-mastoid foramen, 48

- **Paralysis**, bilateral facial, in bilateral lesion of cerebrum, 46
    - of pons, due to disease of basilar artery, 46
  - in cold or double otitis, 46
  - in diphtheritic multiple neuritis, 47
  - in lesion of pons, 46
  - in progressive bulbar paralysis, 46
  - in syphilis at base of brain, 46
  - in toxic peripheral neuritis, 46
- bulbar, 46
  - acute, ophthalmoplegia in, 475
- cerebral, acute infantile, causing hemiplegia, 148
  - deformity of foot in acute, 110
  - diagnosis between paralysis of leg in, and poliomyelitis, 104
    - between hereditary spastic paralysis and, 104
    - between spinal paralysis and, 105
  - epileptic convulsions in, 104
  - gait of infantile, 99
  - mirror-writing in, 82
  - movements of hand in, 81
  - spastic paraplegia in, 103
- crossed, 42
- diphtheritic, loss of knee-jerk in, 627
- due to myelitis, 114
- due to tumor of brain, 536
- Erb's, 87
- family periodic, 125
- glosso-labio-pharyngeal, 46
  - bilateral atrophy of tongue in, 159
  - biting the tongue in, 157
  - cardiac feebleness coming on suddenly in, 318
  - cough in, 599
  - diagnosis of, 633
  - dysphagia in, 164
  - faulty movements of tongue in, 160
  - fibrillary tremor of tongue in, 162
  - indistinct speech of mumbling character in, 633
- hereditary spastic, 104, 105
- infantile spinal. *See* Poliomyelitis, anterior.
- Klumpke's, 88
- laryngeal, causes of, 632
- oculo-facial, 47
- **Paralysis** of bladder with retention, due to pressure during childbirth, 403
- of diaphragm, causes of, 600
  - cough in, 600
- of intraocular muscles, 482
- of muscles of jaw, 42
- of ocular muscles, 467
  - causes of conjugate lateral, 480
  - due to acute or chronic lesions of nuclei, 469, 475
  - due to basilar lesions, 470
  - due to hemorrhage of cerebral cortex, 475
  - due to lesion of nerve-trunks, 470, 476
    - between cortex and nuclear origin of nerve, 470, 476
  - due to poisoning, 469, 475
  - due to sclerosis of cerebral cortex, 475
  - Maddox's rod-test for, 468
    - method of determining individual muscles involved in, 470
    - method of determining partial, 468
  - significance of conjugate, lateral, 480
  - signs of, 470
  - symptoms of thrombosis of cavernous, signs causing, 480
  - varieties of, location of lesions in, 476
- of salivary glands, causing dryness of mouth, 39
- of tongue, 160
  - from injury to hypoglossal fibres outside the medulla, 161
  - from insular sclerosis, 161
  - in diphtheria, 161
  - in paralysis of facial nerve, 161
  - location of lesion in, 161
- progressive bulbar, 47
  - causing paralysis of muscles of mastication, 42
  - diagnosis between asthenic bulbar paralysis and, 48
- pseudo-bulbar, movements of tongue in, 160
- recurrent, of oculomotor nerve of one side, 46
- sometimes found instead of occupation-spasm, 76
- unilateral facial, 38
  - absence of trophic changes in cerebral, 39
  - appearance of face when smiling in, 28

Paralysis, unilateral facial, associated with monoplegia in acute anterior poliomyelitis, 42
  caused by exposure to cold, 38
    by fracture at base of skull, 39
    by hemorrhage from cerebellum, 39
    by hysteria, 39, 41
    by inflammation of nerve-sheath at stylomastoid foramen, 38
    by injuries, 38
    by locomotor ataxia, 39
    by ostitis media, 38
    by pressure of forceps, 39, 40
      of growth at base of skull, 39
    by swelling of parotid gland, 39
  difficulty of whistling and pronouncing labial sounds in, 38
  in crossed paralysis, 42
  in syphilitic arteritis causing other paralyses, 42
  location of cause of, 39
  nerve-degeneration in peripheral, 39
  peculiar distribution of, in some cases, 40
  power of closing eye and wrinkling forehead remains in cerebral form of, 39
  prognosis of, when due to pressure by forceps, 40
  reactions of degeneration in, caused by tumor involving facial fibres below or destroying facial nucleus, 39
Paralytic chorea, 77
  squint, 467
Paramyoclonus multiplex, 78
    diagnosis of, 574
Paramyotonia, 78
Paraphasia, 634
Paraplegia, 102
  ataxic, 98
    knee-jerk exaggerated in, 628
  causes of, 103
  hysterical, knee-jerk exaggerated in, 628
  non-spastic, in acute transverse myelitis, 115
    in chronic transverse myelitis, 116
Paraplegia, non-spastic, in compression from fracture or dislocation of vertebræ, 113, 123
    in diabetes mellitus, rarely, 124
    in hemorrhage into the spinal cord or its membranes, 111, 114
    in hereditary spastic paralysis, 104
    in injury of the cord or myelitis, triple phosphates in urine of, 427
    in Landry's paralysis, 113, 114
    in locomotor ataxia, 114, 122
    in neuritis, 124
    in poliomyelitis, 114
    in polyneuritis, 124
    in subacute transverse myelitis, 116
    in tumor of cord or membranes, 123
    partial, following diphtheria, 124
    pseudo-, in rickets, 124
      in scorbutus in infancy, 124
  of rickets, 103
  reflex, 114
  spastic, 100, 103
    in arrested development, 103
    in rickets, 105
    in spinal lesions, 105
      pachymeningitis, 107
      syphilis, 108
    plantar reflex in, 629
  spinal lesions causing, 103
Parasites, anæmia due to ankylostomum duodenale, 385, 463
    due to tapeworm, 385, 463
  intestinal, ankylostomum duodenale, 462
    ascaris lumbricoides, 462
    bothriocephalus latus, 462
    oxyuris vermicularis, 462
    tænia cucumerina, 462
      mediocanellata, 462
      solium, 460
    trichocephalus dispar, 463
  of blood, 389
  of lung, distoma pulmonum, 605
    echinococcus, 605
  of malaria, æstivo-autumnal, 392
      quartan, 391, 394
      tertian, 390, 394
  of urine, distoma hæmatobium, 412
    filaria sanguinis hominis, 398
Paresis, general, delusions of grandeur in, 404
    cyanosis in, 181
    diagnosis of coma of, 555
    diplopia a symptom of, 466
    fine intention-tremor in, 95
    gait of, 94

Paresis, general, hemiplegia in, 148
Jacksonian epilepsy in, 558
spasm of tongue in, 162
tremor of hands in, 81
vesical symptoms of, 404
Paretic dementia, bilateral atrophy of tongue in, 159
greasy yellow skin in, 178
hemiplegia in, 148
hemorrhages into skin in, 184
localized sweating in, 205
tache cérébrale in, 185
Parkinsonian visage, 32, 573
Parkinson's disease, 79
Paronychia, 59
Parotitis in pyæmic fever, 57
in typhoid fever, 57, 511
in typhus fever, 57
in ulcerative endocarditis, 511
Parrot tongue, 154
Patellar reflex, 625
Pectoriloquy in tuberculosis, 284
over cavity connected with a bronchial tube, 279
Peliosis rheumatica, 182. *See* Purpura.
Pemphigus, eruption of, 200
Peptonuria, 435
Percussion, direct, 258
of heart and great vessels, 264
of respiratory organs, 260
resonance, cause of, 259
trimanual, 347
Percussion-note, coin-test in hydropneumothorax, 288
cracked-pot sound, 261
dull, in consolidation of lung in pneumonia, 263
in pulmonary congestion, 286
œdema, 286
flat, over liver, 348
over pleural effusion, 259
method of eliciting, 258
normal, 259
tympanitic, in lung, due to collapse or adhesion of lung, 261, 263
due to consolidation, 263
due to large cavity, 261, 263
due to pneumothorax, 261
Perforation, leucocytosis in, during typhoid fever, 389
of bowel, pain in, 618
Pericardial friction sounds, 311
Pericarditis, adhesive, symptoms of, 351
pain in, 614
with effusion, diagnosis of, from mediastinal tumors, 297
bulging of chest in, 246
displaced apex-beat in, 256
signs and symptoms of, 267
Pericardium, symptoms of adherent, 255
Perimeter, 490
Perinephritic abscess, 346
Peristaltic waves, 322
Peritonitis, acute, tongue in, 154
character of pain in, 329, 618
drawing up of limbs to relieve pain in, 329
expression of anxiety in, 29, 32
fever in, 585
hiccough in, 576
indicanuria in, 415
muscular rigidity in, 329
position of body and head in, 20
pulse in, 363
retraction of abdominal wall with pain in, 322, 618
small, wiry pulse in, 363
symptoms of acute, 329, 341, 618
tympanites in, 322
vomiting in, 585
Peroneal leg-type of progressive muscular atrophy, 129
Pes equinus, 109
Petechiæ from drugs, 184
in acute ulcerative endocarditis, 184
in cerebro-spinal meningitis, 184
in scurvy, 184
in septicæmia, 184
in snake-bite, 184
in typhus fever, 512
in wasting, 184
Petit mal. *See* Epilepsy, minor.
Phantom tumor of abdomen, 347
Pharyngitis associated with stomach-cough, 599
dysphagia due to, 164
simple acute, not to be confounded with scarlet fever or measles, 167
symptoms of acute, 167
Phenylhydrazin test for sugar in urine, 437
Phlebitis, fever in, 517, 519
Phlegmasia alba dolens, 208
œdema in, 208
Phloroglucin-vanillin test, 339
Phosphaturia, cloudy urine in, 408
headache associated with, 535
Phthisis. *See* also Tuberculosis.
alar chest of, 245
cause of night-cough in, 599
character of cough in laryngeal, 598
of sputum in, 602
"cog-wheel" breathing as an early sign of, 273
diminished expansion of chest in, 284
facial expression in acute, 31
fibroid, physical signs in, 284
flushing of face in, 184, 185
greasy, yellow skin in some cases of, 178
hands in chronic, 58
laryngeal, whispering voice in, 631

- Phthisis, night-sweats of, 204
  - tongue in advanced, 154
  - vomiting in, 593
- Pigeon breast, 247
- Pigmentation of skin, local, 178
- Piles. *See* Hemorrhoids.
- Pin-point pupil in opium-poisoning, 465
- Pitting a sign of dropsy, 206
- Plantar reflex, 629
- Pleural effusion. *See* Effusion, pleural.
- Pleurisy, acute, character of pain in, 286
    - diminished chest expansion in, 253
    - friction-sound often best heard in axilla in, 276, 286
    - pain in, 24, 614
    - position in, 21
    - symptoms and signs of, 286
    - tenderness over rib in, 239
  - hemorrhagic, 290
    - in acute infectious diseases, 290
    - in aneurism, 291
    - in carcinoma, 290
    - in nephritis, 290
    - in tuberculosis, 290
  - interlobular, physical signs of, 291
  - with effusion, 286
    - ægophony above effusion in, 279, 288
    - Baccelli's sign above effusion in, 278
    - bronchial breathing due to compression in, 273
    - bulging of chest in, 246, 287
    - changes in level of effusion in, 286
    - convulsions during irrigation of pleura in, 570
    - cough during aspiration of fluid in, 598
      - on change of position in, 598
    - cracked-pot sound in, 261, 264
    - decrease of vocal fremitus in, 256
      - resonance in, 279
    - diminished chest expansion in, 253
    - displacement of apex-beat in, 257, 288
    - distant breath-sounds over effusion in, 288
    - Litten's sign of, 253
    - method of testing for cause of, 290
    - œdema over ribs in purulent, 209
    - of left side, obliteration of Traube's semilunar space in, 288
      - spleen displaced downward in, 358
- Pleurisy with effusion of right side, liver pushed down in, 352
    - position in, 21
    - pulsating, 255, 258
    - sigmoid line marking top of effusion in, 287
    - skodaic resonance above effusion in, 263, 287
    - symptoms and signs of, 286, 287
- Pleuritis. *See* Pleurisy.
- Plumbic gout, 133
- Pneumococcus as a cause of empyema, 290
- Pneumonia, acute catarrhal, character of fever in, 526
      - difficulty of diagnosis between pulmonary tuberculosis and, 280, 282
      - physical signs of, 282
      - symptoms and signs of, 280
    - character of fever in, 525
    - croupous, casts of finer bronchial tubes sometimes found in sputum of, 604
  - bronchial breathing in, 280
  - bubbling râles in resolution in, 275
  - character of crying in children with, 24
  - Cheyne-Stokes respiration in, 252
  - chill in, 501, 525
  - chlorides decreased in urine in acute stages of, 445
    - increased, indicating convalescence from, 445
  - chronic, Charcot-Leyden crystals in sputum of, 603
  - cracked-pot sound in, 264
  - crepitant râle an early sign of, 275
  - crisis on third to ninth day in, 280, 526
  - croupous, peptonuria in, 435
  - Curschmann's spirals in sputum of, 603
  - delirium a bad symptom of, 280
  - diagnosis and symptoms of, 280
    - between mediastinal growths and, 297
    - between pneumonic phthisis and, 280
  - diminished expansion of chest in, 253
  - expression of anxiety in some cases of, 29
  - face of, 31
  - feeble heart a bad symptom in, 280
  - great increase in urine in convalescence from, 408
  - headache in early stages of, 541, 545
  - herpetic blisters about mouth, with fever in, 31, 199, 280
  - increase of vocal fremitus in, 256
    - resonance in, 278

- Pneumonia, jaundice in, 176
  - leucocytosis in, 388
  - necessity for examining axilla and septum between upper and middle lobe of right lung in, 280
  - percussion-note dull in, 263, 280
  - prune-juice or purulent sputum a bad symptom late in, 280, 601
  - pseudo-crisis in, 280
  - râle redux in, 280
  - rapidity of respiration in, 251
  - rusty, sticky sputum in, 280, 600
  - secondary diphtheritic dysentery due to, 456
  - subnormal temperature following crisis of, 530
  - sweat at crisis in, 204
  - vocal resonance in, 278
  - wavy breathing of, 254
- Pneumonic phthisis, acute, 280
- Pneumothorax, 291
  - amphoric breathing rarely heard in, 274
  - bulging of chest-walls in, 246, 291
  - cracked-pot sound in, 261, 264
  - decrease of vocal fremitus in, 256
  - physical signs of, 291
  - tympanic note in, 261
- Pohl and Rosenbach's test, 290
- Poikilocytes, 368
- Poikilocytosis in chlorosis, 385
  - in pernicious anæmia, 385
- Poisoning, acute alcohol, symptoms of, 548
  - anæmia in chronic lead and arsenic, 385
  - anæsthesia in toxic peripheral neuritis, caused by arsenic, lead, alcohol, and mercury, 230
  - appearance of tongue in acid or alkali, 155, 156
  - Argyll-Robertson pupil in bisulphide of carbon, 484
  - atrophy of tongue in chronic lead, 159
  - auto-intoxication, 533
  - blue line on gums in lead, 163
  - brachial monoplegia in lead, 88
  - cannabis indica, diagnosis of, 549
  - chloral, symptoms of, 550
  - chronic lead, paralyses in, 76
    - silver, 178, 179
  - constipation in phosphorus, 449
  - contraction of pupil in opium, 549
  - convulsions due to aconite, veratrum viride, hydrocyanic acid, ignatia, strychnine, sabadilla, chronic lead, 517
  - diplopia in ptomain, spigelia, conium, belladonna, gelsemium, 466
  - fatty casts in urine due to phosphorus, arsenic, antimony, iodoform, 422
- Poisoning, fever in septic, 519, 522
  - greenish urine in salicylic acid, 414
  - hæmoglobinuria in mushroom, coal-tar, potassium chlorate, glycerin, 412
  - hyperæsthesia in lead and arsenic, 239
  - inflammation of Steno's duct in sulphuric acid, 165
  - jaundice in phosphorus, 173
    - in any poisoning causing excessive hæmolysis, 175
  - joint-affections in chronic lead, 132
  - light-colored stools in phosphorus, 591
  - mercurial, tremors in, 78
  - neuralgia of head in lead and arsenic, 611
  - obstinate constipation in chronic lead, 450
  - ocular paralysis in ptomain, alcohol, sulphuric acid, nicotin, lead, and carbonic acid, 475
  - œdema in arsenic, 207, 208
  - papillitis in alcohol and lead, 497
  - paræsthesia in aconite and carbolic acid, 236
  - peptonuria in phosphorus, 435
  - pin-point pupil in opium, 465, 493
  - pruritus in lead and opium, 239
  - ptosis in gelsemium and conium, 45
  - skin in malarial, 178
  - slowness of breathing in opium, chloral, aconite, chloroform, antimony, 251
  - symptoms of acute antimonial, 452
    - of arsenical, 452
    - of opium, 549
    - of phosphorus, 591
  - tremors of hand in chronic alcohol, lead, and mercurial, 80
  - urine in santonin and carbolic acid, 414
  - vomiting in antimonial, phosphorus, and arsenical, 586, 591
  - wrist-drop in lead, 76, 80
- Poliomyelitis, absence of vesical symptoms in, 404
  - acute, ophthalmoplegia in, 476
  - character of fever in, 529
  - claw-hand in, 72
  - convulsions, headache, and muscular twitching in, 529
  - crural monoplegia, 110
  - diagnosis between paralysis of leg in, and acute cerebral paralysis, 127
  - foot-drop in, 126
  - gait in acute, 98
  - lateral wrist-drop in, 76
  - loss of knee-jerk in, 627
    - of power in several muscles of limb later in disease in, 529

Poliomyelitis, paraplegia in, 109
  "Punchinello" leg in, 127
  symptoms of, 104, 571
Polycythæmia, due to congenital cardiac disease in children, and phosphorus poisoning, 367
Polydipsia, a symptom of diabetes mellitus, 438
Polyneuritis, differential diagnosis between paraplegia in, and Landry's paralysis, 109
Polynuclear leucocytes, 382
Polyphagia a symptom of diabetes mellitus, 438
Position as an encouraging sign, 21
  due to action of drugs, 20
  in acute articular rheumatism, 20
    bellyache, 20
    nausea, 20
    pleurisy, 21
    asthma, 21
  in chronic bronchitis, 21
  in coma, 20
  in dyspnœa, 21
    due to asthma, 21, 293
  in emphysema, 21
  in enlarged liver, 21
  in grave or advanced disease, 21
  in heart disease, 20
    severe, 21
  in hemorrhage, external or internal, 20
  in large growths in abdominal cavity, 21
  in œdema of lungs, 21
  in peritonitis, 20
  in pleurisy with effusion, 21
  in pulmonary consolidation, 21
  indicative of convalescence, 20
  persistently on the side, 21
Post-hemiplegic chorea, 147
Potassium ferrocyanide test for albumin in urine, 432
Pot-belly, 322
Pott's disease, 105
    spastic paraplegia in, 105
Pregnancy, caries of teeth in, 163
  chloasma of, 178
  discomfort due to, 22
  grave prognosis of acute diffuse nephritis in, 446
  hemorrhagic retinitis in, 499
  pigmentation of eyelids during, 28
  value of estimation of urea during, 444
  vomiting in, 590
Pressure sensibility of skin, 212
Presystolic mitral murmur, 303
Prevost's symptom, 480
Proctitis, symptoms of, 454, 455
Progressive bulbar paralysis, bilateral facial paralysis in, 46
Prostate, enlarged, a cause of interference with passage of urine, 406
Prune-juice sputum in pneumonia, 280, 601
Pruritus about anus from opium, 239
  in chronic lead-poisoning, 239
  in contracted kidney, 239
  in diabetes, 239, 447
  in gout, 239
  in jaundice, 174, 240
  in piles, 240
Pseudo-angina pectoris, pain in, 613
  -bulbar paralysis, diagnosis of, 47, 634
  -leukæmia, diagnosis of, 388
  -membranous dysentery, 456
  -meningitis, convulsions in children in, 370
  -muscular hypertrophy, gait in, 98, 99
  -ptosis, caused by paralysis of the unstriped fibres of Müller, 44
  -tabes, 87, 91
    gait in, 91
Psilosis, 157
Psoriasis, nails in, 59
Ptosis, alternate, in so-called nervous syphilis, 44, 46
  associated with internal squint, 45
  caused by affection of the sympathetic, 44
    by cerebral hemorrhage, 44
    by disease of the corpora quadrigemina, 45
    by lesion of oculomotor nerve or nucleus, 42
      in angular gyrus, 44
    by nervous syphilis, 44
    by reflex irritation, 44
  complicating tetanus, 44
  concomitant symptoms of, if oculomotor nerve is destroyed, 43
  congenital, 42, 45
  definition of, 42
  in feeble, overworked women in the morning, 45
  in hysteria, 44
  in idiopathic muscular atrophy, 44
  in locomotor ataxia, 45
  in meningitis, 579
  in oculo-facial paralysis, 47
  in recurrent paralysis of oculomotor nerve on one side, 46
  in tubercular or syphilitic changes at base of brain, 45
  transient, in poisoning by gelsemium or conium, 45
  with hemiplegia of face and limbs on opposite sides of the body, 45
Puerile breathing, 272
Puerperal eclampsia, diagnosis of convulsions in, 568

Puerperal melancholia, lingual spasm in, 162
Pulmonary abscess, 285
    sputum in, 602
  congestion, 286
  consolidation, position in, 21
  disease in children, pain in, 23
  gangrene, 286
    breath in, 19
    vomiting in, 594
  œdema, 286
    position in, 21
  osteo-arthropathy, appearance of nails in, 59
    enlargement of feet in, 128
    spade-like hands in, 68
  resonance, 259
  stenosis, 309
Pulsating pleurisy, 255, 258
Pulsation, epigastric, 343
  of retinal arteries in aortic regurgitation, 313
  on chest-wall, 254
    causes of, 254
Pulse, best taken in children when asleep, 25, 359
  blood-vessels and, 359
  causes of rapid, 366
    of slow, 366
  Corrigan, 363
  dicrotic, 364
  examination of, 359
  feeble, rapid, and running, in ruptured compensation of heart muscles, and in cholera morbus, 303, 451
  force of, 361, 365
  hard and tense, in acute diffuse nephritis, 446
  high tension, in acute fevers, 362
      in chronic interstitial nephritis, 362, 447
      in parenchymatous nephritis, 362
  in acute alcohol-poisoning, 548
  in chloral-poisoning, 550
  in opium-poisoning, 549
  in uremic coma, 550
  low tension, in exhausting diseases, 363
      in mitral stenosis, 363
      in ruptured compensation, 363
  method of counting, 360
    of production of, 360
  normal wave of, 360
  of empty arteries in severe hemorrhage, 365
  pulsus alterans, 364
    paradoxus, 363
  rapid and weak, in intestinal obstruction, 582
Pulse, rapid, due to stimulation of heart, 366
  rate of, 363
  slow in cerebral tumor, 537, 578
    of digitalis, 363
  tonometer of Gaertner to study tension of, 362
  trip-hammer, 363
  volume of, 363
  water-hammer, 305, 313, 363
  -wave, normal, 364, 365
"Punchinello" leg, 127
Pupil, Argyll-Robertson, 484
  contracted, 465
    from central nervous disease, 465
    from cerebral inflammation, 465
      tumor, 485
    from chronic tobacco-poisoning, 485
    from corneal inflammation, causing photophobia, 465
    from locomotor ataxia, 485
    from meningitis, 465, 485
    from paretic dementia, 485
  contraction and dilatation of, with Cheyne-Stokes breathing, 486
  dilated, 465
    from blindness, 465
    from fright, 465
    from glaucoma, 465, 485
    from hysteria, 465
    from intracranial pressure causing coma, 465
    from irritation of cervical sympathetic, 486
      of upper part of spinal cord, 485
    from mydriatic, 465
    from paralysis of centre of third nerve, 485
  hippus, 486
  immobile, 465, 484
  irregular and sluggish in syphilis, 186
  method of testing, 483
  pin-point, in opium-poisoning, 465, 549
  reaction of, to accommodation, 483
    to light, 483
  size of normal, 483
  transitory, unequal dilatation of, in tuberculosis, 486
  Wernicke's sign of hemiopic inaction of, 486, 537
Purpura hæmorrhagica, 183
    hæmatemesis in, 587
    hæmaturia in, 412
    hæmoptysis in severe, 601
    Henoch's disease in, 183
    in rheumatism, 182
    joint-involvment with fever in, 528

Pus-casts in urine, 420
Pustular syphiloderm, 196
Putrid bronchitis, 292
Pyæmia, chill in, 501
diazo-reaction in severe, 445
jaundice in, 176, 178
joint-involvement with fever in, 528
parotitis in, 57
profuse sweating in, 204
Pyæmic abscess of liver, 353
Pyelitis, character of fever in, 448, 520
of pain in, 448, 621
chills and fever in, 448, 520
diagnosis of, 448, 621, 622
may be caused by strongylus gigas, 412
pain in lumbar region in, 448
pyuria constant or intermittent in, 448
urine acid in, 448
Pyelonephrosis, casts of micrococci in, 421
Pylephlebitis, vomiting in, 592
Pyopneumothorax, coin percussion in, 288
Hippocratic succussion in, 279
metallic tinkling in, 275
subphrenicus, 342

QUINCKE'S SIGN in aortic regurgitation, 313

RAILROAD BRIDGE tremor, 81
Râles, 274
altered by coughing if not crepitant, 275
bubbling, in bronchitis, 275
in convalescence from asthmatic attack, 275
from pneumonia, 275
in pulmonary œdema, 275
in resolution in pneumonia, 275
in tuberculosis, 275, 283
character of, in asthma, 293
crepitant, in breaking down of tubercular lung tissue, 275
in early stages of croupous pneumonia, 275
in pulmonary collapse or œdema, 275
congestion, 286
redux, in pneumonia, 280
sonorous, 274
Rash of scarlet fever, 186
Raynaud's disease, appearance of hands in, 59
coma in, 554
gangrene in, 67, 201
hæmoglobinuria in, 413
localized sweating in, 205
Raynaud's disease, symptoms of, 201, 202
vomiting in, 591
Rectal catarrh, acute symptoms of, 454, 455
Rectum, obstruction of, 585
Reduplication of heart-sounds, 299
Reflex iridoplegia. *See* Argyll-Robertson pupil.
Reflexes, ankle clonus, how elicited, 626
biceps tendon, how tested, 626
cremasteric, 626
chin-jerk, how elicited, 626
diseases in which decreased, 627
in which increased, 628
knee-jerk, how best elicited, 625
reinforced, 625
loss of ocular, in acute alcoholic poisoning, 548
of skin, table of, 629, 630
plantar, 629
scapulo-humeral of von Bechterew, 626
tendon, 625
Regurgitation, aortic, 304
pulse in, 363
mitral, 300
pulse in, 363
tricuspid, 309
Relapse, frequency of, in Malta fever, 512
in typhoid fever, 507
Relapsing fever, character of fever in, 512
hæmatemesis in, 587
hyperæsthesia in, 238
jaundice in, 175
œdema of legs and wrists in, 209
spirillum of Obermeier in blood in, 513
sweating in, 204
tongue in, 152
Remittent fever. *See* Malaria, remittent.
Renal colic, retraction of abdominal wall in, 322
disease, puffiness under eyes in, 28, 32
Resonance, vocal, 277
Baccelli's sign, 278
decreased, causes of, 278
increased, causes of, 278
Respiration, causes of labored, 251
of rapid, 251
of slow, 252
Cheyne-Stokes, 252
costal, 253
counting, 250
diaphragmatic, 253
in chloral-poisoning, 550
in coma of apoplexy, 552
in diabetic coma, 551
in healthy child, 24
in uræmic coma, 551

- Respiration, inspiration prolonged in spasmodic croup, 252
  - method of studying, in children, 25
  - prolonged expiration in asthma and emphysema, 252
  - ratio of pulse to, 251, 252
  - rhythm of, 251, 252
  - stertorous, in acute alcohol-poisoning, 548
    - in opium-poisoning, 549
  - wavy, in pneumonia, 254
- Retention-cysts of pancreas, 341, 342
  - of urine in locomotor ataxia, 404
    - in myelitis, 405
    - in overdistention, 405
    - in pressure during childbirth, 405
- Retina, œdema of, in general arterial disease, 499
- Retinitis, 498
  - albuminuric, 499
  - hemorrhagic, 499
  - in diabetes, 499
  - in leukæmia, 388
  - in syphilis, 499
- Retraction of head in children, causes of, 52
  - of ribs in adherent pericardium, 255
- Retro-bulbar neuritis, 497
- Retro-œsophageal abscess sometimes causes dysphagia, 164
- Rheumatic form of gonorrheal arthritis, 130
- Rheumatism, acute articular, appearance of hands in, 66
    - of tongue in, 153
  - character of fever in, 528
  - chlorides decreased in, 445
    - increased in, in convalescence, 445
  - convulsions in, 528
  - cyanosis in, 528
  - erythema in, 182
  - headache in, 535
  - in the knee or ankle, 130
  - iritis in, 499
  - joint-affections in, 528
  - papillitis in, 497
  - purpura in, 182
  - spontaneous dislocation of the hip following, 134
  - subcutaneous fibrous nodules in, 185
  - sweating of, 205
  - uric acid in urine of, 425
  - urticaria in, 185
  - with hyperpyrexia, delirium in, 528
  - chronic, changes in shape of hand in, 65
    - limping gait in, 90
  - of scalp, symptoms of, 612
- Rheumatoid arthritis. *See* Arthritis deformans.
- Rhonchi, 274
- Ribs, systolic retraction of, in adherent pericardium, 255
- Rickets, beaded ribs of, 249
  - carpo-pedal spasm in, 75
  - cranio-tabes in, 53
  - deficient bony development in, 53
  - early decay of teeth in, 163
  - excessive sweating of head in, 56
  - gait in, 99, 102
  - Harrison's grooves in, 247
  - head in, 30, 52
  - hyperæsthesia in, 239
  - laryngeal spasm in, 294
  - nodding spasm in, 50
  - open fontanelle in, 53
  - pain in back in, 615
  - pseudo-paraplegia in, 124
  - spastic paraplegia in, 105
  - subperiosteal hæmatoma in, 132
  - sweating of head in, 205
  - tenderness of skin in, 239
- Risus sardonicus in epilepsy, 556
  - in tetanus or strychnine-poisoning, 28, 49
- Roberts's yeast-test for sugar in urine, 438
- Romberg's symptom, 92
- Röntgen rays in diagnosis of tuberculosis, 285
- Roseola. *See* also Erythema.
  - due to antidiphtheritic serum, 190
  - due to drugs, 192, 193, 194
  - in Bright's disease, 191
  - in chickenpox, 189, 198
  - in cholera, 191
  - in diabetes, 192
  - in diphtheria, 189
  - in German measles, 188
  - in malaria, 189
  - in septicæmia, 190
  - in smallpox, 189, 194
  - in syphilis, 190
  - in tubercular peritonitis, 192
  - in typhoid fever, 189, 191
  - in typhus fever, 189
  - in vaccination, 189
- Rose rash. *See* Erythema and Roseola.
- Rötheln, appearance of rash in, 188
  - fever in, 514
  - symptoms of, 188
- Rubella. *See* Rötheln.
- Rubeola. *See* Measles.

- SACCHAROMYCES albicans, causing fermentation in diabetic urine, 430
- St. Vitus's dance. *See* Chorea.
- Salaam convulsions, 573
- Salivary calculus, 165
- Saltatoric spasm, 78, 574

Saprophytic organisms as a cause of empyema, 290
Sarcinæ ventriculi, in vomitus from chronic gastric catarrh, 589
      from gastric cancer, 589
        dilatation, 589
        ulcer, 589
      test for, 589
Sarcoma, anæmia due to, 385
  enlargement of liver in, 349
  purpura in multiple, 180, 184
Scalp, rheumatism of, pain in, 612
"**Scaphoid**" belly, 322
Scapulo-humeral reflex of von Bechterew, 626
    exaggerated in neurasthenia, 626
Scarlet fever, arthritis in, 133
    character of fever in, 513
    Cheyne-Stokes respiration rare in, 252
    chill in, 501
    collar of brawn in, 513
    date of eruption in, 189
    diazo-reaction in severe, 445
    enlargement of spleen in, 356
    erythema in, 186
    hæmaturia in, 409
    lysis in fever of, 513
    otitis in, 513
    pleurisy with effusion complicating, apt to become purulent, 289
    prognosis from fever, 513
    rose rash in, 186
    sore throat in, 166
    splenic enlargement in, 356
    spontaneous dislocation of the hip following, 134
    strawberry tongue in, 154
    synovitis in, 67
Scars of the skin, 203
Schönlein's disease, joint-involvement in, 133
    symptoms of, 183
    ulcerations of tongue in, 157
    ulcer of buccal mucous membrane in, 165
Sciatica. *See* Neuritis, sciatic.
Sciopody, 129
Sclerema neonatorum, symptoms of, 211
Sclerodactyle, nails in, 59
Scleroderma, skin in, 37, 210
Sclerosis, amyotrophic lateral, 73
      alterations of hands in, 73
      ankle-clonus in, 630
      character of speech in, 634
      knee-jerk increased in, 628
      spastic paraglegia in, 105
  disseminated, allochiria in, 236
      ankle-clonus in, 630
Sclerosis, disseminated, coma in, 555
      convulsions in, 570
      differential diagnosis of, 97, 484
      expression of face in early stages of, 32
      gait in, 99
      hemianæsthesia in, 218
      hemiplegia resulting from, 148
      hippus in, 486
      knee-jerk increased in, 628
      nystagmus in, 482
      optic atrophy in, 497
      paralysis of ocular muscles in, 475
        of tongue in, 161
      slow, scanning speech in, 633
      spasm of tongue in, 162
      symptoms of, 80
      tremor of hands in, 79
        of head in, 80
      vertigo in, 547
  general arterial, accentuation of heart-sounds in, 298
  lateral, absence of vesical symptoms in, 404
      ankle-clonus in, 630
      causes of increased reflexes in, 106, 107
      gait in, 98
      spastic paraplegia in, 105
  multiple cerebro-spinal, 106
Scorbutus. *See* Scurvy.
Scotoma, 497
Scrofulosis, 35
  abdomen in, 322
  dactylitis in, 59
  tongue in, 154
Scurvy, corneal ulceration in, 499
  dropsy in, 207
  ecchymosis under the eyes in, 28
  hæmatemesis in, 587
  hæmaturia in, 412
  loosening of teeth in, 164
  pain in back in, 615
  petechiæ in, 184
  pseudo-paralysis in, in infancy, 124
  sponginess of gums in, 164
Seal-fin hands in arthritis deformans and gout, 65
Senile chorea, 77
Senility, tremor of hands in, 79
Sensation, disturbances of, 235
Sensibility, disturbances of, 211
  method of testing, 212
        tactile, 212
        thermic, 213
  perverted, 216
Septic arthritis, 132
Septicæmia, blood in, 359
  character of fever in, 519, 524
  Cheyne-Stokes respiration rare in, 252

- Septicæmia, diarrhœa in, 456
  - great variations in temperature in, 524
  - jaundice in, 175
  - petechiæ in, 184
  - retinal hemorrhages in, 499
  - rose rash in, 189
  - splenic enlargement in, 358
- Serous effusions, sugar in, 326
  - pleural effusion, cause of, 290
    - Pohl and Rosenbach's test to differentiate from effusion due to inflammation, 290
- Sero-purulent effusions, absence of sugar in, 326
- Shiga's bacillus, epidemic dysentery due to, 456
- Singultus, 576
  - causes of, 576
- Skin, anæsthesia in, 216
  - cadaveric, in pyæmia, 178
  - cold and moist, with high rectal temperature, in sunstroke, 528
    - and wet, a bad symptom in fever, 504
  - color of, in health and disease, 169
  - cyanosis of, 180
  - dropsy and swelling of, 206
  - dry and hot, in fever, 504
  - eruptions of, in disease, 182
  - excess of dryness of, 205
  - gangrene of, 201
  - glossiness of, 200
  - greasy-yellow in phthisis and chronic liver disease, 178
  - greenish-yellow in chlorosis, 178
  - harsh and dry, in acute diffuse nephritis, 446
    - in cholera, 205
    - in diabetes, 205, 206, 447
  - hemorrhages into, 184
  - hyperæsthesia of, 237
  - in acute alcohol-poisoning, 548
  - in Addison's disease, 179
  - in angioneurotic œdema, 211
  - in argyria, 178, 179
  - in carcinoma of internal organs, 178, 180
  - in chloasma of pregnancy, 178
  - in elephantiasis, 210
  - in influenza, 239
  - in jaundice, 170
  - in kidney disease, 180
  - in myxœdema, 210
  - in œdema neonatorum, 210
  - in opium-poisoning, 239, 549
  - in profound anæmia, 239
  - in sclerema neonatorum, 211
  - in scleroderma, 210
  - in tuberculosis, 179
- Skin, increased sensibility of, due to ergot, 239
  - leucoderma, in true goitre, 179
  - muddy-yellow, in chronic malaria and prolonged suppuration, 178
  - pain of, 239
  - pallor of, 180
    - in myxœdema, 180
  - paræsthesia of, 235
  - parchment-like, in syphilis, 28
    - pigmentation of, 178, 321
    - by the prolonged use of arsenic, 179
    - due to scratching, 179
  - pruritus of, 239
  - redness of, 185
  - scars of, 203
  - sensation of, 211
  - sweating of, 204
  - table of reflexes of, 630
  - *tache cérébrale*, 185
    - *meningeale*, 185
  - tenderness of, in diseases of internal organs, 239
    - in rickets, 239
  - waxy, in cretinism, 34
  - yellow, in xanthoma, 177
    - and greasy, in paretic dementia, 178
- Skodaic resonance, 263, 287
- Smallpox, appearance of eruption in, 189, 194, 514
  - date of eruption in, 189, 194
  - fever in, 514
  - grisolle sign of, 189
  - hæmoptysis in hemorrhagic, 601
  - headache in early stage of, 545
  - pain in back in onset of, 514, 624
  - spasms, 572
- Smokers' patch on tongue, 158
- Snake-bite, purpuric eruptions following, 184
- Sonorous râles, 274
- Sounds, heart-, reduplication of, 299
- Spasm, habit-, 48
  - hysterical, of œsophagus, 165
  - nodding, 50. *See* Nodding spasm.
- Spasms, 572
  - blepharo-facial, 48
    - Graefe's spots in, 48
  - carpo-pedal, 75
  - facial, Abadie's sign in, 48
    - cause of functional, 48
      - of organic, 48
    - following exposure to cold, 50
      - paralysis, 50
    - in chorea, 48
    - in hysteria, 49
  - in adults, due to locomotor ataxia, 294
    - due to mediastinal growths, 294
  - in children, due to croup, 294

- Spasms in children due to digestive disturbances, 294
  - due to laryngeal catarrh, 294
  - due to rickets, 294
  - in chorea insaniens, 573
    - minor, 572
  - in electric chorea, 573
  - in epilepsy, 48
  - in Huntingdon's chorea, 573
  - in lesion of middle peduncle of cerebellum, 575
  - in meningitis, 48
  - in paramyoclonus multiplex, 574
  - in tetanus, 48, 51
  - in Thomsen's disease, 575
  - in true chorea, 572
  - laryngeal, in adults, due to aneurism pressing on recurrent laryngeal nerve, 294
    - due to enlargement of retro-bronchial glands, 295
  - of fingers due to occupation, 75, 76
  - of tongue, 162
  - saltatoric, 574
  - varieties of, 572
- Spastic infantile hemiplegia, 73
  - monoplegia, 108
  - paraplegia, 103. *See* Paraplegia, spastic.
- Specific gravity method of estimating hæmoglobin, 379
  - of urine, 417
- Speech, 631. *See* also Voice.
  - defects of, 634
    - agraphia, 634
    - alexia, 635
    - aphasia, 634
    - aphemia, 634
    - aphonia, 635
    - apraxia, 635
    - conduction aphasia, 635
    - paraphasia, 634
    - tests for, 635
    - word-blindness, 635
    - word-deafness, 635
  - feeble, hesitating, in pneumonia of pulmonary œdema, 632
  - halting, hesitating, in paretic dementia, 633
  - incoherent, in chorea in children, 633
    - in delirium, 633
  - indistinct, mumbling, in amyotrophic lateral sclerosis, 634
    - in glosso-labio-pharyngeal paralysis, 633
    - in stomatitis, 633
    - in typhoid fever, 633
  - nasal, in adenoid vegetations, 632
  - nervous, mechanism of, 634
  - scanning, in multiple sclerosis, 99
  - shrill, piping, in paralysis agitans, 634
- Speech, slow, scanning, in disseminated sclerosis, 633
    - in Friedreich's ataxia, 633
  - tests for defects of, 635
  - whispering, causes of, 631
- Spermatorrhœa, 429
  - accompanying oxaluria, 426
  - spermatozoa found in, 429
- Spermatozoa in urine, significance of, 428, 429
- Sphygmograph, 364
- Spinal cord, great rise in temperature due to injuries to cervical portion of, 529
  - localization of functions of segments of, 119
  - location of lesion of, in anæsthesia, 224
  - loss of knee-jerk in injuries of, 627
- Spine, caries of, symptoms of, 346
- Spirillum of Obermeier in blood in relapsing fever, 513
- Spleen, causes of enlargement of, 358
    - of extension downward of, 358
  - enlarged, in amyloid disease of kidney, 447
    - in leukæmia, 388
    - in typhoid fever, 356
  - floating, 346
  - method of examination of, 355
  - normal position of, 355
  - swelling of, in septicæmia, 358
    - in Weil's disease, 520
  - uric acid in excess in enlargement of, 425
- Splenic anæmia, 356
    - blood changes in, 356
  - fever. *See* Anthrax.
- Spontaneous dislocation of the hip, after infectious diseases, 134
- Spots of Valleix, 613
- Spotted fever. *See* Cerebro-spinal meningitis.
- Sputum, actinomyces in, 605
  - anchovy sauce, in amœbic abscess, 602
  - blood-streaked, in leakage from aortic aneurism, 601
    - in pulmonary gangrene in children, 601
    - in valvular heart disease, 601
  - bloody, diagnosis between bloody vomit and, 601
    - in hæmoptysis of aortic aneurism, 601
    - in ruptured blood-vessels in posterior pharyngeal wall, 601
    - of hæmophilia, 601
    - of hemorrhagic smallpox, rarely, 601
    - of pulmonary tuberculosis, 601

- **Sputum**, bloody, of severe purpura, 601
    - of valvular cardiac disease, 601
    - of vicarious menstruation, 601
  - brick-dust, in hepatic abscess communicating with lung, 602
  - brownish, in gangrene of lungs, 286
  - casts of bronchioles in, in fibrinous bronchitis, 604
    - of larynx and bronchi in, in diphtheria, 603
    - of smaller bronchioles found occasionally in, in croupous pneumonia, 604
  - Charcot-Leyden crystals in asthmatic, 294, 603
  - copious and purulent, in pulmonary abscess, 285
  - crystals of margaric acid in pulmonary gangrene, purulent bronchitis, and follicular tonsillitis, 605
  - Curschmann's spirals in asthmatic, 294, 603
  - Dittrich's plugs in, in purulent bronchitis, 292
  - eggs of distoma pulmonum in, 605
  - elastic fibres in, in abscess of lung, 605
      - in gangrene of lung, 605
      - in tuberculosis of lung, 605
  - examination of, for elastic fibres, 604
    - with centrifuge, 604
  - frothy, in pulmonary œdema, 286
  - hooks of echinococcus in, 605
  - in asthmatic attacks, 602
  - in chronic bronchitis, 292
  - in pulmonary gangrene, 286, 602
  - liquid and watery, in epidemic influenza, 602
      - in Bright's disease, 602
      - in pulmonary œdema, 602
  - macroscopic examination of, 600
  - method of staining for tubercle bacilli in, 606
  - microscopical examination of, 604, 606
  - prune-juice, in chronic pneumonia, 280, 601
  - purulent, causes of, 602
  - rusty, sticky, in croupous pneumonia, 600
  - semi-liquid, in pulmonary phthisis, 602
  - thick and yellowish, in later stages of acute bronchitis, 600
- **Squint**, diagnosis between concomitant and paralytic, 467
    - of conditions producing, 473
  - external and internal, due to tumor at base of brain, 536
    - causes of, 477
  - in hysteria, 481
  - in meningitis, 579
- **Squint** in ocular palsy, 45, 470
  - internal, causes of, 477
  - significance of external, 477
- **Staphylococci** as a cause of empyema, 290
- **Status epilepticus**, 565
- **Stellwag's** symptom, 465
- **Stenosis**, aortic, 304
    - pulse in, 363
  - mitral, 300
    - pulse in, 363
  - pulmonary, 309
- **Steppage** gait, 91
- **Stereognosis**, 213
- **Stomach**, cancer of, 332
  - cough due to reflex irritation from, 596
  - dilatation of, 334
    - vomiting in, 588
  - distention of, by gas, 335
  - indicanuria in cancer of, 415
  - method of examining, with gastrodiaphane, 336
      - with gyromele, 336
  - palpation and percussion of, 328
  - test of contents for hydrochloric acid, 338, 339
    - for lactic acid, 338
    - for urates in urine in disorders of, 425
  - ulcer of, 333
  - vomiting in dilatation of, 588
- **Stomatitis**, action of child with, toward breast, 24
  - appearance of tongue in ulcerative, 157
  - buccal mucous membrane affected in, 165
  - epidemic, symptoms of, 527
  - impetiginosa, 159
  - malignant ulcerative, 165
  - offensive odor of breath in, 19
  - temperature of mouth raised in, 503
- **Stone** in bladder, interference with passage of urine due to, 406
- **Stools**. *See* also Feces.
  - almost devoid of mucus in cholera morbus, 451
  - bilious, 459
  - bloody, 459, 584
  - clay-colored, in hepatogenous jaundice, 175
    - in Weil's disease, 520
  - gallstones in, 459
  - in cholera, comma bacilli in, 452
    - infantum, 451
  - in dysentery, 455
  - in intussusception in children, 580, 581
  - in proctitis, 454, 455
  - intestinal parasites in, 462
  - mixed with mucus, 459
  - oily, in carcinoma of pancreas, 334

- Stools, pus in, 459
  - ribbon-shaped, in follicular enterocolitis, 454
  - rice-water, in antimonial poisoning, 452
  - significance and character of blood in, 457, 459
  - tarry, in yellow fever, 521
- Strabismus. *See* Squint.
- Strawberry tongue, 154
- Streptococci as a cause of empyema, 290
- Stricture of œsophagus. *See* Œsophageal stricture.
- Strongylus gigas, hæmaturia due to, 412
- Stupor in typhoid fever, 509
  - in ulcerative endocarditis, 511
- Subdural hemorrhage, 553
- Subperiosteal hæmatoma, 132
- Subsultus tendinum, 60, 81
- Succussion, Hippocratic, 279
  - in pleural effusions with pneumothorax, 288
- Sucking movements in meningitis, 541
- Sudamina, 205
- Sugar in the blood, 400
    - Williamson's test for, 400
  - in the urine. *See* Glycosuria and Urine.
- Sunstroke, character of fever in, 528
  - diagnosis of coma in, 552, 555
  - headache of, 545
  - skin cold and moist with high rectal temperature in, 528
    - hot and dry in, 528
- Suppurative tonsillitis, symptoms of, 166
- Swallowing, difficult. *See* Dysphagia.
- Sweat, bile-stained, in jaundice, 205
- Sweating in acute articular rheumatism, 205
    - miliary tuberculosis, 525
    - ulcerative endocarditis, 204
  - in antimonial poisoning, 452
  - in collapse, 204
  - in crises in fevers, 204
  - in debility, 205
  - in health, 204
  - in hepatic abscess, 517, 519
  - in intermittent malarial fever, 204
  - in malaria, 204
  - in obstruction of rectum, 585
  - in phthisis, 204
  - in pneumonia at crisis, 204
  - in pyæmia, 204
  - in relapsing fever, 204
  - in typhoid fever, 204
  - in uræmia, 205
  - localized, 205
    - in exophthalmic goitre, 205
    - in hysteria, 205
    - in migraine, 205
    - in paretic dementia, 205
    - in Raynaud's disease, 205
- Sweating, localized, in rickets, 205
    - in thoracic aneurism, 205
  - uriniferous, in deficient renal activity, 205
- Symmetrical gangrene, 201, 202
- Syncope, diagnosis of, 569
- Synovitis, acute, 130
- Syphilis, action of child with, toward breast, 24
  - alternate ptosis in, 44, 46
  - aortitis in, 613
  - appearance of fingers in dactylitis in, 59
    - of nails in, 59
  - Argyll-Robertson pupil in cerebral, 484
  - associated with amyloid kidney, 447
  - at base of brain causing bilateral facial paralysis, 46
  - atheroma in, 361
  - broadness of bridge of nose in congenital, 30
  - diagnosis between cerebro-spinal syphilis and disseminated sclerosis, 99, 485
    - of coma in, 554
    - of epilepsy due to, 560
  - diagnosis of, by studying the state of hæmoglobin, 386
  - early cutting of teeth in inherited, 163
  - enlargement of spleen in early, 356
    - of tongue in, 159
  - eruptions of, 190, 196, 203
  - features in congenital, 30
  - fever in, intermittent, 526
    - remittent, 526
    - simple continued, 526
  - fissures and chancres of tongue in, 156
  - gummatous swelling and periosteal thickening of shins in, 135
  - hemiplegia in cerebral, 148
  - Hutchinson teeth in, 163
  - immobility of pupil in, 484
  - iritis in, 499
  - irregular pupil in, 186
  - Justus's sign of, 381
  - miscarriage a symptom of, 22
  - mucous patches about mouth and anus in infantile, 30
  - of intestinal tract, diarrhœa due to, 456
  - open fontanelle in, 53
  - pain in periostitis of skull in, 612
  - papillitis in, 497
  - paræsthesia in spinal, 236
  - parchment-like skin of, 28
  - pseudo-palsy in, 88
  - ptosis in changes at base of brain in, 44
  - purpura in, 184

Syphilis, purulent acne of forehead in, 196
  retinitis in, 499
  rose-rash in, 186, 190
  scars in, 204
  spastic paraplegia in spinal, 108
  symptoms of arteritis due to, 538
  ulceration of tongue in, 156
Syphilitic arteritis, 538
    paralyses in, 42
  epilepsy, 560
  gumma, 538
  meningitis, 538
  periostitis of skull, pain in, 612
  rupia, 203
  triad, 163
Syphiloderm, pustular, 196
Syringomyelia, appearance of nails in, 59
  loss of pain and temperature-sense in, 223
  necrosis of terminal phalanges in, 68
  partial anæsthesia in, 223
  patches of anæsthesia in, 223
  small joints affected in, 129
  symptoms of, 224
  wasting of hands with anæsthesia in, 73

TABES dorsalis *See* Locomotor ataxia.
  Tabetic atrophy, 497
    foot, 129
    ulcer, 135
Table of reflexes, 630
Tache cérébrale, 185
  méningéale, 185
Tachycardia, 316, 317, 366
Tactile sensibility of skin, 212
Tænia cucumerina, 462
  mediocanellata, 462
  solium, 460
Tallquist method of estimating hæmoglobin, 381
Tapeworm. *See* Parasites; Tænia.
Taste, decrease of acuity of, in facial hæmiatrophy, 37
  loss of, in anterior portion of tongue in disease of petrous portion of temporal bone, 39
Teeth, ages at which different, appear in children, 163
  blue line around, in lead-poisoning, 163
  caries of, in diabetes mellitus, 163
    in pregnancy, 163
  cut early in inherited syphilis, 163
  early decay of, in rickets, 163
  grinding of, in children, 164
  Hutchinson, 163
  loosening of, in salivation, 164
    in scurvy, 164
  staining of, 163
Teichmann's crystals in hæmoglobinuria, 413
Temperature. *See* also Fever.
  causes of sudden fall of, in typhoid fever, 506
  great rise of, in injuries to cervical cord, 529
  high, in cholera infantum, 527
    in sunstroke, 528
    with cold skin in cholera Asiatica, 527
  in acute alcohol-poisoning, 549
  in diabetic coma, 551
  in intestinal obstruction, 583
  in uræmic coma, 550, 551
  methods of taking, 501
  subfebrile, mildly febrile, decidedly febrile, 501
  subnormal, causes of, 530
    in algid type of pernicious malaria, 530
    in cretinism, 35
    in heat-exhaustion, 530
    in injuries to dorsal portion of spinal cord, 530
Tendon-reflexes, 625
Tenesmus in bladder-trouble, 402
  in cholera infantum, 451
  in dysentery, 455
  in intussusception, 581
  in proctitis, 454
  in stricture of rectum, 453
Tension, asynchronous of sigmoid valves causing reduplication of second sound of heart, 299
Terminal leucocytosis, 389
Test, coin-, in hydro-pneumothorax, 288
  Ehrlich's diazo-reaction, 445
  Esbach's, for albumin in urine, 433
  for acetone in urine, 439
  for albumin in urine, boiling, 430
      Esbach's, 433
      Heller's, 431
      potassium ferrocyanide, 432
      quantitative, by centrifuge, 432
      trichlor-acetic acid, 432
  for bile in urine, 414, 440
  for chlorides, 444
  for chyle in urine, 415
  for defects, intellectual, 635
  for elastic fibres in lungs, 604
  for hæmin crystals, 594
  for hydrochloric acid in stomach, 338, 339
  for indicanuria, 415, 440
  for lactic acid in stomach, 338
  for peptone in urine, 435
  for sarcinæ ventriculi, 589
  for sensibility of skin, 212
  for stricture of œsophagus, 164
  for sugar in urine, Fehling's, 436

- Test for sugar in urine, Haines's, 435
    - Roberts's, 437
    - phenyl-hydrazin, 437
    - Trommer's, 437
    - Whitney's, 435
  - for tubercle bacilli, 606
  - for urates, 414
  - for urea, Lyons's, 440
  - Gabbett's modification of Fränkel's method for tubercle bacilli, 608
  - Gmelin's, for bile in urine, 414, 440
  - guaiacum, for hæmoglobin in urine, 413
  - Haines's modification of Haessar's method for total quantity of solids in urine, 417
  - Heller's, for blood in urine, 413
  - methylene-blue, to determine activity of kidneys, 418
  - of Maddox, rod-, 468
  - of pupils, 483
  - Oppenheimer's modification of Denigès', for acetone in urine, 439
  - Stokvis' modification of Jaffé's, for indican in urine, 440
  - Widal's, for typhoid fever, 400
  - Williamson's, for sugar in the blood, 400
- Tetanus, character of convulsions in, 570
    - of fever in, 528
  - facial spasm in, 48, 51
  - head or cephalic, 51
  - knee-jerk increased in, 628
  - ptosis in, 44
  - risus sardonicus in, 28
- Tetany, "accoucheur's hand" in, 75, 571
  - character of convulsions in, 571
  - Chvostek's symptom of, 571
  - Erb's symptom of, 571
  - gastric dilatation in, 75
  - laryngismus stridulus in, 571
  - thyroid wasting in, 75
  - Trousseau's symptom of, 571
- Thermic fever. *See* Sunstroke.
  - sensibility of skin, 212
- Thoma-Zeiss's hæmatocytometer, 370
- Thomsen's disease, diagnosis of, 575
    - spasm of hand in, 79
      - of tongue in, 162
- Thorax, aneurism of, diagnosis between medicinal growths and, 295
    - localized sweating in, 205
    - spongy, venous masses above clavicle due to, 253
  - inspection of, 241
- Thrill, causes of, 258
  - diffuse and feeble in cardiac dilatation, 313
  - in aortic aneurism, 305
  - in carotid arteries, 317
  - in hydatid cyst of liver, 353
- Thrill in mitral regurgitation in children, 302
    - stenosis, 303
- Thrombosis, cerebral, 74
    - brachial monoplegia in, 85
    - coma in, 553
    - œdema in, 209
    - of lateral sinus, Gerhardt's symptom of, 544
    - of superior longitudinal sinus, 542
  - of cavernous sinus, 543
      - symptoms of, 543
  - of coronary artery, 318
  - of femoral artery, 134
  - of lateral sinus, 544
      - localized œdema in, 209
  - of umbilical vein in melæna neonatorum, 587
  - of vena azygos, as a cause of serous pleural effusion, 290
  - vomiting in, 577
- Thrush, appearance of tongue in, 153
- Thyroid wasting in tetany, 75
- Tic, convulsive, 78
    - facial spasm in, 48
- Tinnitus aurium in Ménière's disease, 592
- Tobacco-amblyopia, 497
  - -heart, 316
- Toe, tender, in sciatica, 616
- Toes, separation of, in ainhum, 135
- Toison's solution for counting white corpuscles, 373
- Tongue, anatomy of the, 150
  - annulus migrans of, 159
  - appearance of, in acute articular rheumatism, 153
      - catarrhal jaundice, 153
      - gastric catarrh, 153
          - in children, 153
          - inflammation of, 159
    - in Addison's disease, purple spots on, 155
    - in advanced disease of exhausting nature, 154
    - in anæmia, 155
    - in biliousness, 152
    - in cardiac disease, 155
    - in cretinism, 34
    - in dysentery, 154
    - in grave disease of the viscera, 154
    - in hemiplegia, 154
    - in hepatic abscess, 154
    - in mucous disease, worm-eaten, 153
    - in persons taking large amounts of milk, 153
    - in poisoning by acids, 155
      - by alkalies, 156

Tongue, appearance of, in poisoning by cantharides, 156
by corrosive sublimate, 155
in pulmonary disease, 155
in relapsing fever, 152
in scarlet fever, strawberry, 154
in scrofulosis, 154
in stomatitis impetiginosa, 159
in syphilis, 156
in thrush, 153
in tonsillitis, 151, 153
in typhoid fever, in typhoid state, 152
in ulcerative stomatitis, 157
in uræmia, 152
bilateral atrophy of, due to disease affecting hypoglossal nerve, 159
in chronic lead-poisoning, 159
in glosso-labio-pharyngeal paralysis, 159
in locomotor ataxia, 159
in paretic dementia, 159
in progressive muscular atrophy, 159
biting of, in epilepsy, 157
in glosso-labio-pharyngeal paralysis, 157
chancre of, 156
chronic superficial glossitis of, 158
coating of, causes of, 151
composition of, 151
unilateral, due to decayed teeth or hemiplegia, 154
dryness or moisture of; prognostic value of, 154
enlargement of, in acromegaly, 160
in actinomycosis, 160
in acute inflammation, 159
in inflammatory hypertrophy, 159
in macroglossia, 159
in myxœdema, 160
in new growths, 159
in syphilis, 159
epithelioma of, 156
eruptions of, 157
examination of, 150
in children best when crying, 25
fibrillary tremor of, in labio-glosso-pharyngeal paralysis, 162
fissures of, due to age, chewing tobacco, or abuse of alcohol or tea, 156
deep, in dissecting glossitis, 156
geographical, 159
glossodynia exfoliatava of, 158
hemiatrophy of, 160
ichthyosis of, 158
leucokeratosis of, 158
leucoma of, 158
Tongue, lichen planus of, 158
movements of, 160
in coma, 160
in feeble-minded, 160
in glosso-labio-pharyngeal paralysis, 160
in nervous individuals, 160
in pseudo-bulbar paralysis, 160
in typhoid fever, 160
mucous patches of, 156
mycosis of, 153
œdema of, 159
paralysis of, 160
in diphtheria, 161
in injury of hypoglossal or trifacial nerve, 161
location of lesion in, 161
parrot, in dysentery or hepatic abscess, 154
patches on, in chronic superficial glossitis, 158
in leucoma, 158
in smokers, 158
purple spots on. in Addison's disease, 155
scleroderma of, 158
smoker's patch on, 158
spasm of, 162
in aphthongia, 162
in chorea, 162
in general paralysis of the insane, 162
in hysteria, 162
insular sclerosis, 162
in irritation of hypoglossal nerve, 162
in puerperal mania, 162
in Thomsen's disease, 162
stains of, 153, 154, 155
strawberry, in scarlet fever, 154
tremor of, in alcoholism, 162
in glosso-labio-pharyngeal paralysis, 162
in typhoid fever, 162
ulceration of, in epithelioma, 156
in lupus, 157
in psilosis, 157
in Schönlein's disease, 157
in stomatitis, 157
in syphilis, 156
in wounds, 156
multiple, in tuberculosis, 156
of frænum of, in whooping-cough, 157
unilateral atrophy of, in chronic lead-poisoning, 159
urticaria of, 158
wandering rash of, 159
worm-eaten, in mucous disease, 153
xanthelasma of, 155
xeroderma pigmentosum of, 158
Tonometer of Gaertner, 362

Tonsillitis, breath in, 19
  chronic night-cough in, 599
  crystals of margaric acid in follicular, 605
  diagnosis between diphtheria, scarlet fever, and, 166
  dysphagia in, 164
  fever in, 527
  odor of breath in, 18
  symptoms of follicular, 166
    of suppurative, 166
  tongue in, 151, 153
Tonsils, chronic cough in enlarged lingual, 599
  night-cough in enlarged faucial, 599
  pain in calculus of, 168
  symptoms of chronic enlargement of, 168
Torula cerevisiæ causing fermentation in gastric dilatation, 589
Tracheal tugging in aneurism, 308
Transitional leucocytes, 381
Traube's semilunar space obliterated in left-sided pleural effusion, 288
Tremor of hands and arms, 79
    in mercurial poisoning, 80
    railroad-bridge type of, 81
  of tongue, 161
    in alcoholism, 162
    in glosso-labio-pharyngeal paralysis, 162
    in typhoid fever, 162
  of type Rendu, 81
  post-hemiplegic, 81
  sometimes found instead of occupation-spasm, 76
Trichiniasis, diagnosis of, 512
  leucocytosis in, 382, 388
  puffy eyelids in, 32
Trichinosis. *See* Trichiniasis.
Trichlor-acetic acid test for albumin in urine, 432
Trichocephalus dispar, 462, 463
Tropical abscess of liver, 353
Tricuspid regurgitation, 309
  stenosis, 309
Trimanual percussion, 347
Trip-hammer pulse of aortic regurgitation, 305, 313, 363
Trommer's test for sugar in urine, 437
Tropical dysentery, 455
Trousseau's symptom of tetany, 571
Tube-casts. *See* Casts.
Tubercle bacilli, method of examining and staining, 606–608
Tubercular glands, symptoms of, 340
  meningitis, 540
    differential diagnosis of, 540
    headache in, 540
    papillitis in, 497
    in children, 541
  pyelitis, 448
Tuberculosis, amphoric breathing in, 274, 284
  anæmia in, 385
  as a cause of empyema, 290
  bronchial breathing and consolidation in, 272, 273
  bronchitis a cause of death in, 22
  bronchophony in, 284
  bubbling râles in, 275, 283
  Charcot-Leyden crystals in sputum of, 603
  Cheyne-Stokes breathing in meningeal, 252
  chronic loose morning-cough in cavity in, 597
  cracked-pot sound in, 263
  crepitant râles and breaking down of lung tissue in, 275
  cyanosis in acute miliary, 525
  dactylitis in, 60
  diagnosis between catarrhal pneumonia and pulmonary, 282
  diarrhœa of, 454
  diazo-reaction in severe pulmonary, 445
  elastic fibres in sputum of, 605
  expiration prolonged in, 284
  fever in acute miliary, 525
    pulmonary, 524
  frog-belly in, 322
  hæmoptysis in, 601
  heavy face in bone disease in, 30
  hectic fever in, 284
  hemorrhagic pleurisy in, 290
  hollow-chested build in, 19
  leucocytosis in, 509
  loss of flesh in, 284
  mammary gland enlarged in, 249
  metallic tinkling in, 275
  multiple ulceration of tongue in, 156
  necessity of examining axilla in, 285
  ocular palsy in, 475
  of bladder, hæmaturia due to, 411
  of bones in amyloid kidney, 447
  of kidney, 411, 621
  of mesenteric glands, 340
  of omentum, 340
  pectoriloquy in, 279, 284
  peptonuria in, 435
  percussion-note dull in consolidation due to, 262
  pleurisy complicating, apt to become purulent, 289
    with effusion coming on insidiously in undiscovered, 290
  ptosis in changes at base of brain due to, 44
  puerile breathing an early sign of, 272
  rapidity of breathing in, 251
  retraction of abdominal wall in, 322
  Röntgen rays in diagnosis of, 285

- **Tuberculosis, scars on neck from suppurating glands in, 204**
  - spirituelle face of children with diathesis of, 30
  - sputum, Curschmann's spirals rarely seen in, 603
  - sweating in, 284
  - tongue in, 154
  - transitory, unequal dilatation of pupils in, 486
  - vocal fremitus and resonance increased in, 278, 283
  - vomiting in acute miliary, 579
  - Wintrich's change of percussion-note in, 284
- **Tubular breathing, 271, 273**
- **Tumor, abdominal, pain in, 622**
  - at base of brain, symptoms of, 40
  - brain-, Cheyne-Stokes breathing in, 252
    - contracted pupil in, 485
    - hyperæsthesia in, 238
    - impaired memory in, 578
    - papillitis in, 496, 578
    - paræsthesia in, 236
    - severe headache in, 578, 612
    - slow pulse in, 578
    - spastic hemiplegia in, 147
    - symptoms of, 536
    - unilateral anæsthesia in, 218
    - vertigo in, 547, 578
    - vomiting independent of taking food in, 578
  - detection of, in intussusception, 581
  - diagnosis of fatty, in abdominal wall, 331
  - in renal pelvis, symptoms of, 621
  - of abdomen, 331, 340
  - of bladder, 406
  - of cord, bladder symptoms of, 404
    - girdle-sensation in, 237
    - paraplegia in, 123
  - of lung, rapid respiration in, 251
  - of mediastinum, 295
  - of nerves, pain in, 624
  - of omentum, 343
  - phantom, 347
  - pontine, anæsthesia in, 220
- **Türck's gyromele, 336**
- **Tympanites in flatulent colic, 322**
  - in intestinal obstruction, 583
  - in peritonitis, 322
  - in typhoid fever, 322, 509
- **Typhoid fever, appearance of eruption in, 191, 509**
  - bacillus of Eberth the cause of, 509
  - blood in, 389
  - bloody stools in, 459, 584
  - bradycardia in, 317
  - carphologia in, 81
- **Typhoid fever, causes of mumbling speech in, 633**
  - of recrudescence of fever in, 507
  - of vomiting in, 585
  - character of coma in, 551
    - of fever in, 506
  - Cheyne-Stokes respiration rare in, 252
  - chill in, 501
  - coma rare in, 551
  - date of eruption in, 189
  - delirium in, 32, 509
  - diagnosis, 509
    - between acute miliary tuberculosis and, 509
    - between Malta fever and, 512
    - between remittent fever and, 510
    - between trichiniasis and, 512
    - between ulcerative endocarditis and, 511
  - Ehrlich's diazo-reaction in, 445
  - enlargement of spleen in, 356, 509
  - face in, 31
  - headache in, 545
  - hyperæsthesia in convalescence from, 238
  - hypertrophic osteo-arthritis following, 133
  - in children, 520
  - increase of urine in convalescence from, 408
  - lesions of lungs generally at base in, 509
  - leucocytosis absent unless perforation occurs during, 389, 509
  - meteorism in, 509
  - moderate bronchitis early in, 509
  - mushy stool in, 458
  - œdema of thigh over deep muscular abscess following, 209
  - pain in, 619
  - parotitis in, 511
  - peptonuria in, 435
  - phlegmasia alba dolens in, 208
  - pleurisy with effusion complicating, apt to become purulent, 289
  - recrudescence in, 507
  - relapse in, 507
  - roseola in, 189, 191, 509
  - splenic enlargement in, 356
  - spontaneous dislocation of the hips following, 134
  - stools in, 509
  - stupor in, 509

- Typhoid fever, subsultus tendinum in, 81
  - sudden fall in temperature due to hemorrhage of any sort in, 506
    - due to intestinal hemorrhage in, 506
    - due to intestinal perforation in, 506
  - sweating in, 204
  - tongue in, 152
    - movements of, 160
    - tremor of, 162
  - tympanites in, 322, 509
  - vomiting in, 585
  - Widal's test for, 400, 509
  - without fever, 507
- Typho-malarial fever. *See* Malaria, remittent.
- Typhus fever, character of fever in, 512
  - crisis in, 512
  - eruption in, 191, 512
  - great exhaustion in, 512
  - hæmatemesis in, 587
  - headache in, 512

- ULCER of stomach, symptoms of, 333
    - vomiting in, 586
  - perforating, 134
  - tabetic, 135
- Ulcers at base of finger-nails due to chloral, 60, 202
  - on buccal mucous membrane in Schönlein's disease, 165
  - on cornea in scrofula, 499
  - on tongue, 156
  - perforating, in diabetes, senility, and tabes dorsalis, 134
  - syphilitic, in intestinal tract causing diarrhœa, 456
- Umbilical hernia, 328, 347
- Unconsciousness. *See* Coma.
- Unilateral anæsthesia, 218
  - facial paralysis, 38. *See* Paralysis, unilateral facial,
  - reflex iridoplegia, significance of, 484
- Uræmia. *See* also Coma.
  - amaurosis in, 500
  - anæmia in, 385
  - breath in, 19
  - Cheyne-Stokes breathing in, 252
  - colliquative diarrhœa in, 577
  - coma in, 550
    - occurring in chronic interstitial nephritis, 447
  - convulsions in, 577
    - diagnosis of, 567
  - delirium, mild, 480
  - headache in, 535
- Uræmia, hiccough in, 576
  - sweating in, producing scales of urea, 205
  - symptoms of, 550
  - tongue in, 152
  - vomiting in, 551, 577
    - odor of carbonate of ammonium in, 577
- Ureometer, 441
- Urethra, character of blood in hemorrhage from, 411
  - fever due to passage of sound into, 505
  - gonococci in specific inflammation of, 428
  - incontinence of urine due to insensitive, 405
  - interference with passage of urine in stricture of, 406
- Urethritis, specific, gonococci in, 428
- Uricæmia. *See* Lithæmia.
- Uridrosis, 205
- Urine, acetone in, 439
    - Oppenheimer's modification of Dennigés' test for, 439
  - acid fermentation of, 429
  - albumin in, boiling test for, 430
    - Esbach's method of testing for, 433
    - Heller's test for, 431
    - potassium ferrocyanide test for, 432
    - quantitative test for, by centrifuge, 432
    - trichlor-acetic-acid test for, 432
  - albuminuria, significance of, 434
  - albumose in, 434
    - in Bradshawe's disease, 434
    - in normal puerperium, 434
  - alkaline fermentation of, 430
  - bile in, 440
  - bile-stained, in hepatogenous jaundice, 175
  - blood in, 409
  - casts in, blood, 409, 420
    - epithelial, 419
    - fatty, 422
    - granular, 421
    - hyaline, 422
    - of micrococci, 421
    - of pus, 420
  - causes of incontinence of, 405
    - of retention of, 402
  - chlorides in, significance of increase and decrease of, 445
    - test for, 444
  - color of, blue, 415
    - brown, 414
    - causes of black, 409
    - due to blood, 409
    - green, 414
    - in diabetes, 415

- Urine, color of, in jaundice, 414
    - red, 409, 414
    - table of changes in, 416
    - white or milky, 415
    - yellow, 414
  - conditions in which decreased, 408
    - in which increased, 408
  - crystals in, creatin, 426
    - creatinin, 426
    - oxalate of lime, 425
    - triple phosphates, 426
    - urate, 425, 426
    - uric acid, 425
  - cylindroids in, 422, 423
  - diazo-reaction of Ehrlich in, 445
  - embryos of parasites in, 428
  - epithelial cells in, 428
  - fermentation of, 429, 430
  - Gmelin's test for bile in, 414, 440
  - gonococci in, 428
  - guaiacum test for hæmoglobin in, 413
  - Heller's test for blood in, 413
  - in acute diffuse nephritis, 445
  - in amyloid kidney, 447
  - in cholera morbus, severe, decreased in 451
  - in chronic interstitial nephritis, 446
  - in diabetes insipidus, 447
    - mellitus, 447
  - in intestinal obstruction, 583
  - in phosphorus-poisoning, 591
  - in pyelitis, 448
  - in uræmia, 550
  - indican in, 440
    - test for, 440
  - interference with passage of, due to enlarged prostrate, 406
      - due to nervous bladder, 406
      - due to stone in bladder, 406
      - due to stricture of urethra, 406
      - due to tumors of bladder, 406
  - involuntary passage of, in apoplexy, 406
      - in idiots, 405
      - in infectious diseases, 406
      - in irritation of foreskin or vagina in children, 406
      - in seat-worms, 406
      - in some cases of insanity, 406
  - method of collecting, for testing, 407
  - microscopic examination of, 418
  - odor of, causes of alteration of, 408
  - peptone in, 435
    - test for, 435
  - specific gravity of, causes of high, 417
      - causes of low, 417
  - spermatozoa in, 428, 429
- Urine, staphylococci and streptococci in, indicating pus, 428, 429
  - Stokvis' modification of Jaffé's test for indican in, 440
  - sugar in, Fehling's test for, 436
    - Haines's test for, 435
    - phenyl-hydrazin test for, 437
    - quantitative yeast-test of Roberts for, 438
    - significance of, 438
    - Trommer's test for, 437
    - Whitney's test for, 435
  - total quantity of solids in Haines's modification of Haesser's method for computing, 417
  - tubercle bacilli in, 428
  - urates in, 425, 426
  - urea in, amount excreted in health, 444
    - causes of increase and decrease of, 444
    - Lyon's method of estimating, 440
    - value of estimating in renal disease during pregnancy and before surgical operations, 444
  - uric acid in, 425
  - variations in quantity of, 407
- Urobilin icterus, 176
- Urticaria, 192
  - from drugs, 185
  - in rheumatism, 185
  - of tongue, 158

- VACCINATION, appearance of eruption of, 196, 197
  - roseola following, 189
- Vaccinia, 196
  - eruption in, 196, 197
- Vaginitis, specific, gonococci in, 428
- Valvular heart disease. *See* Heart.
- Varicella. *See* Chickenpox.
- Variola. *See* Smallpox.
- Vena azygos, thrombosis of, as a cause of serous pleural effusion, 290
- Vertigo, 547
  - causes of, 547
  - due to drugs, 547
  - in Addison's disease, 593
  - in anæmia, 384, 547
  - in aortic obstruction, 312
  - in apoplexy, 547
  - in brain-tumors, 547, 578
  - in disseminated sclerosis, 547
  - in epilepsy, 547
  - in indigestion, 547
  - in ingravescent apoplexy, 144
  - in Ménière's disease, 547
  - in middle-ear disease, 547
  - in ocular palsy, 470
  - in paretic dementia, 148
  - in pernicious anæmia, 384

Vertigo in phosphorus-poisoning, 582
  paralyzing, 547
Vesical crises in locomotor ataxia, 92
Vesicular breathing, 271
Vision, changes in acuity of, 487
  failure of, 487
Vitiligo, 180
Vocal fremitus. *See* Fremitus, vocal.
  resonance. *See* Resonance, vocal.
Voice, 631
  hoarseness of, 631
  in aneurism, 631
  in paralysis agitans, 634
  in typhoid fever, 633
  loss of, or mutism, 632
  nasal, causes of, 632
  whispering, causes of, 631
Volvulus, 343, 584
  vomiting caused by, 584
Vomit, 594
  of chronic gastric catarrh, 588, 594
  of gastric cancer, 333, 586, 588
    dilatation, 588
    ulcer, 586
  of uræmia, 551, 577
  significance of, 594
  test for hæmin in, 594
    for sarcinæ in, 589
Vomiting, 576
  associated with headache, 593
  bilious, in remittent malarial fever, 520
  black, in yellow fever, 521
  centric, from chloroform or ether, 576
  coffee-ground, in gastric cancer, 332, 586
    in locomotor ataxia, 586
    in phosphorus-poisoning, 591
  cyclical, 590
  decrease of urine by persistent, 408
  due to injuries to the epigastrium, 587
  in acute gastric catarrh, 588
    miliary tuberculosis, 579
    pancreatitis, 591
    yellow atrophy of liver, 593
  in Addison's disease, 593
  in appendicitis, 585
  in brain-tumor, 536, 578
  in catarrhal or obstructive jaundice, 592
  in cerebellar tumor, 578
  in cerebral disease, 577
  in cerebro-spinal meningitis, 522
  in cholera Asiatica, 585
    infantum, 451, 586
    morbus, 451, 585
  in chronic gastric catarrh, 588
  in diabetes, rarely, 577
  in dilatation of the stomach, 588
  in dysentery, 586
Vomiting in early secondary syphilis, 594
  in embolism of superior mesenteric artery, 591
  in enterocolitis, not common, 453
  in erysipelas, 594
  in exophthalmic goitre, 592
  in gastric cancer, 586
    ulcer, 586
  in gastro-pulmonary fever, 530
  in hæmoglobinuria, 593
  in hemiplegia, due to hæmatoma of dura mater, 148
  in hemorrhagic infarction of the intestine, 584
    pressure on brain, 147
  in hepatic abscess, 592
    cirrhosis, 587
  in hepatitis, 592
  in hysteria, 590
  in influenza, 525
  in ingravescent apoplexy, 144
  in intestinal obstruction, 579, 582
  in intussusception, 579
  in melæna neonatorum, 587
  in Ménière's disease, 592
  in meningitis, 578
  in merycismus, 594
  in migraine, 593, 611
  in neurasthenia, 590
  in neuroses of stomach, 590
  in obstruction of rectum, 585
  in peritonitis, 585
  in phosphorus-poisoning, 591
  in phthisis, 593
  in poisoning by arsenic and antimony, 452, 586
  in pregnancy, 590
  in profound cerebral anæmia, 578
  in pulmonary gangrene, 594
  in purulent meningitis, 578
  in pylephlebitis, 592
  in Raynaud's disease, 591
  in renal calculus, 593
  in true gastritis, rare, 588
  in typhoid fever, 585
  in uræmia, 576, 577
  in whooping-cough, 594
  in yellow fever, 521, 593
  of blood. *See* Hæmatemesis.
  reflexly produced by gastric, intestinal, or other abdominal disorders, 576, 579
  uræmic, in chronic parenchymatous nephritis, 446
  ushering in eruptive diseases, 594
Von Fleischl's hæmoglobinometer, 376

WANDERING rash, 159
  "Water-hammer" pulse of aortic regurgitation, 305, 313, 363
Wavy breathing in pneumonia, 254

"Waxy" casts in urine, 422
Weil's disease, almost total absence of gastro-intestinal symptoms in, 520
    character of fever in, 520
    clay-colored stools in, 520
    jaundice in, 173, 520
    swelling of liver and spleen in, 520
Wernicke's pupil, 486, 537
Westphal's sign, 92
Whispering voice, 631
White blood-corpuscles, method of counting, 373
  infarct in coronary arteries, 318
Whitlow, painless, in Morvan's disease, 72
Whitney's test for sugar in urine, 435
Whooping-cough, Cheyne-Stokes respiration rare in, 252
  cough in, 596
  hemorrhage of the skin in, 184
  ulcer on frænum in, 157
  vomiting in, 594
Widal's test for typhoid fever, 400, 509
Williamson's test for sugar in blood, 400
Wintrich's change of percussion note in tuberculosis, 284
Word-blindness, 635
  -deafness, 635
Worms, 460
  involuntary passage of urine due to seat-, 406
Wrist-drop, causes of, 76
Wrist-jerk, marked, in amyotrophic lateral sclerosis, 73
Wry-neck, causes of, 50
  clonic or tonic, 50, 51
  congenital or acquired, 50, 51
  definition of, 50
  diagnosis of, 51
  facial asymmetry in connection with, 35

XANTHELASMA, 155
  Xanthoma, 177
Xeroderma pigmentosum, 158

YELLOW atrophy of liver, acute, convulsions in, 572
    coma in, 551
    hæmatemesis in, 587
    jaundice in, 175
    symptoms of, 175, 588, 593
  fever, black vomit in, 521
    character of fever in, 521
    diagnosis of, 521, 522
      between bilious remittent fever and, 521
      between dengue and, 521
    hæmatemesis in, 587
    hemorrhage from mucous membranes in, 521
    jaundice in, 173, 521
    symptoms of, 593
    tarry stools in, 521
    vomiting in, 593
  spot of the eye, 496

ZIEHL'S solution for staining tubercle bacilli, 607

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